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OTO-LARYNGOLOGICA

SUPPLEMENTUM 200

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ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

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FROM THE DEPARTMENT OF OTOLARYNGOLOGY
KAROLINSKA SJUKHUSET AND THE DEPARTMENT OF HYGIENE
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*To Brita*

# Contents

# Introduction

Air pollution has become an increasing problem for the industrialized countries, tobacco smoking is considered to play an important role in the development of carcinoma of the lung and larynx and all over the world research is going on to identify and neutralize the noxious exhaust products from gasoline and oil combustion.

Defending the respiratory mucosa against these and other agents, such as dust, soot and bacteria, the mucous layer covering the epithelium also helps in conditioning the inspired air during its passage to the alveoli. Under normal conditions the greater part of the cleansing and conditioning process is performed by the nasal mucosa.

To accomplish this the surface has to be wiped clean, for otherwise these particles might accumulate and even multiply in the mucous cover. This clearance is brought about by *inter alia* ciliary movement, dragging the viscous layer with its embedded material towards the pharynx, from where it is disposed of by swallowing.

Although cilia were discovered almost 300 years ago, it took another 150 years for them to become the subject of physiological studies in animals. Even today our knowledge is based mainly on animal experiments, while studies on normal human ciliary mucosa are very few in number. This is easily understood when the fact is considered that the human ciliary mucosa is hidden in places almost inaccessible to direct observation without interfering with its normal conditions. Conclusions drawn from animal and excised tissue experiments provide the basis for our present knowledge of human ciliary activity, together with some *in vivo* studies on humans, most of them, however, done under non-physiological conditions.

It is therefore the aim of this study to describe a new method for observing the mucus flow in the human nose under physiological conditions and to present the results from an investigation using that method.

# Basic Morphology and Physiology

The gross anatomy and physiology of the human nose and the histology of the respiratory mucosa are generally accepted facts which need only be briefly recapitulated

The blood supply comes from both the external and internal carotid arteries via the sphenopalatine artery to the turbinates lateral walls and inferior parts of the septum and via the ethmoidal arteries to the superior parts of the nose The capillary network is well developed especially in the turbinates

The sensory nerves come from the ophthalmic and maxillary branches of the trigeminal nerve and the autonomic nerves reach the mucosa almost entirely via the sphenopalatine ganglion through which the postganglionic sympathetic fibres from the first two thoracic segments synapsing in the superior cervical ganglion pass without relaying while the parasympathetic fibres from the pons reach the sphenopalatine ganglion via the facial great superficial petrosal and vidian nerves in order to synapse The sympathetic nerves are considered mainly as vasoconstrictors while the parasympathetic fibres are thought to be vasodilatory and secretory (Malcolmson 1959)

The human nasal mucosa is covered by a so-called pseudostratified columnar ciliated epithelium with mucus producing goblet cells and ciliated cylindrical cells overlying a layer of variable thickness consisting of transitional cells In the submucosa are numerous glands containing serous and mucous cells (Messerklinger 1958 Taylor, 1958)

This so-called respiratory mucosa lines the posterior two thirds of the internal nose except the olfactory region where a special sensory epithelium is found In the anterior third where the non conditioned inspiratory air hits the mucosa and on the anterior ends of septal deviations and turbinates the epithelium is mostly of a non ciliated type (Oppikofer 1907 Hilding 1931c 1932a Dixon *et al* 1949)

The air currents in the nose have been the subject of extensive studies and their normal pattern is not a matter of controversy In man the inspiratory air arches upwards towards the roof and the olfactory region from where it curves downwards towards the nasopharynx Septal ridges spurs and other irregularities of the nose cause *variations but the main pattern is the same* (*Proetz* 1953) This leaves the inferior part of the nose in relative calm with a well developed ciliated epithelium and a subsequent high rate of mucus flow (Hilding 1931a 1932a)

The mucous sheet produced by the glands and goblet cells and covering the epithelium is composed of two layers an outer viscous stratum resting on the tips of the moving cilia and an inner one of lower viscosity which forms a suitable medium for the vibrating cilia (Lucas & Douglas 1934 Breuninger 1964) From the inactive non-ciliated anterior parts of the nose the mucous blanket is slowly pulled by traction from active areas further back In these regions speeds of 10 mm/min have been recorded in humans (Hilding 1931a)

Recent studies with electron microscopy show the ultrastructure of ciliated cells to be almost identical in different animal and even plant species (Engström, 1951, Engström & Wersäll, 1952, Manton, 1952, Fawcett & Porter, 1954, Rhodin & Dalhamn, 1956, Afzelius, 1959, 1960, 1961, *i a*) The cilia, 250—300 on each cell (Rhodin, 1959, Spoendlin, 1959), are approximately 8 microns long and 0 3 microns in diameter, extending from the free end of the columnar cell, with an individual basal body just below the cell surface Cross sections of cilia show nine peripheral double filaments and two centrally located single ones In humans the peripheral filaments pass longitudinally from the tip of the cilium to the basal body, while the central ones terminate just above the level of the cell surface and are thought to have a stabilizing effect, only permitting bending of the cilium in one plane, the peripheral filaments and the basal body are considered to be the motor unit in the ciliary beat The mechanism and chemical process involved is, however, still a matter of dispute (Sleigh, 1962)

# Review of the Literature

Ciliary movement was first described by Antonio de Heide in 1683, according to various authors (Proetz, 1953, Tremble, 1962) William Sharpey gave the complete history of ciliary research before 1835 in his chapter on "Cilia" in Todd's Cyclopaedia of Anatomy and Physiology (1836), from which it could be seen that during those 150 years the main interest was focused on observing ciliary activity in lower animals, such as infusoria, sponges, mussels, etc According to Sharpey it was Otto Frederick Muller who coined the name "cilia" in 1773

During the early 1830s Purkinje and Valentin in Breslau and Sharpey in London independently studied ciliary activity in higher animals Purkinje and Valentin were the first to discover cilia at work in the Fallopian tube of a pregnant rabbit and in 1834 they gave the first report on ciliary activity in the respiratory tract of different reptiles birds and mammals using a pigmented solution as indicator while observing excised strips of epithelium under a microscope (Purkinje & Valentin, 1834)

At the same time Sharpey was studying the respiratory mucosa in dogs and rabbits by applying charcoal powder He found the mucus flow to be directed towards the larynx in the lower airways of the dog and towards the ostium in the maxillary sinus of the rabbit, and made the following conclusion "Ciliary motion at least serves to convey the secretions along the membranes together with foreign matter" (Sharpey, 1836) Sharpey Purkinje and Valentin also reported on the microscopic details, the fanning or lashing movement of the individual cilia, the progressive waves, later called metachronal rhythm, across the ciliated surface, these waves resembled "those produced by the wind in a cornfield, and were quite independent of the will of the animal, continuing long after death" The effects of external agents, e g electricity, temperature, fresh water, acids, and inflammation were also mentioned The reports and conclusions of these authors are cited in some detail because they are remarkably accurate and still valid, although they were made more than a century ago

Some theories on the mechanism of ciliary movement were also advanced at that time According to Sharpey R E Grant considered cilia to be hollow organs which were distended by the injection of fluid into them from a tubular system along their base Purkinje and Valentin observed that the cilia were bulbous at their base and thought it probable that they were moved by a muscular substance in the bulb or around it Sharpey however considered it possible that "cilia may contain muscular substance throughout a greater or less portion of their length, by which they can be bent or extended or perhaps they may in some instances be bent by muscular fibres and resume their original shape and position by virtue of their elasticity" In 1891 Schäfer formulated his theory that ciliary movement was caused by a rhythmic flowing of hyaloplasm into and out of the cilium from the cell while Heidenhain in 1911 considered ciliary activity to be an active process in the cilia themselves by means of

an alternating contraction and relaxation of the surface layer As has been shown, these theories date back to the 1830s and even today the exact nature of ciliary movement is unknown The basal body has been shown to play an important part, as the cilium continues beating as long as it is intact, Peter (1899) was the first to show that cilia completely isolated from the distal protoplasm where the basal body is located, *are motionless*

During the last century basic research on ciliary movement has followed three main paths microscopy, ciliary beat studies and mucus flow studies

## *Microscopy*

Until the 1940s light microscopy did not bring anything particularly new, compared with earlier studies, but with the coming of the electron microscope the ultrastructure of the ciliated cells began to be revealed, with the results already mentioned (Engstrom, 1951, Engström & Wersall, 1952, Manton, 1952, Fawcett, 1954, Fawcett & Porter, 1954, Rhodin & Dalhamn, 1956, Rhodin, 1959, Spoendlin, 1959, Afzelius, 1959, 1960, 1961, Gibbons, 1961, Burian & Stockinger, 1963)

## *Ciliary beat studies*

Aside from simply observing the cilia under a microscope in excised specimens or lower animals, no attempts were made to measure the beat rate until the end of the 19th century, when the stroboscope was introduced The stroboscope technique has more recently been used in beat studies by Krueger & Smith (1957, 1958a, b) and Krueger *et al* (1959) Gray (1930) used a motion picture camera to record the ciliary beat for subsequent counting of the rate, and this photographic technique was further developed by Lucas and Proetz Lucas (1933) introduced reflected light and relatively low magnification for observing the flickering light reflexes caused by groups of cilia beating in approximately the same phase. This technique was well suited for *in vitro* and *in vivo* studies of small animals but could not be used for observing more deeply situated areas By using a vertical "Ultropak' illuminator Proetz (1933) overcame this difficulty and the technique has subsequently been used by a number of other authors Frenckner & Richtner (1939) described a microscope with a sufficiently long working distance for clinical *in vivo* observations and found the ciliary beat rate in the human nose to be 160—250 beats per minute This relatively low rate as compared to those of more recent studies is possibly explained by a less exact recording technique Proetz (1953) gives the normal rate as 480—720, whereas Dalhamn (1956 1960), Dalhamn *et al* (1959), using a high speed motion camera Krueger & Smith (1957 1958a, b), Krueger *et al* (1959), using a stroboscope, and Toremalm (1961) using high speed photography, all give beat rates of 600—1500 irrespective of the techniques or animals used *In vivo* observations on humans under physiological conditions using these modern devices have so far not been feasible, but judging from *in vitro* studies the normal beat rate can be assumed to be around 1000 beats per minute

travelled distance measured The average speed was 4 5 mm per minute in normal cases, 2 2 and 7 3 mm per minute when the dog was under the influence of morphine and caffeine respectively The relatively low average speed may be explained by the findings of Hilding (1932b) that the speed of flow increases towards the ostium of the frontal sinus of a living dog Hilding (1957, 1961b) assumes the existence of such a speed gradient also in the bronchial tree, with reported variations in speed between a medium sized bronchus and the trachea

Florey *et al* (1932) examined the flow of a suspension of graphite ink in the living cat's trachea and found the normal speed to be 35 mm per minute Stimulation of the recurrent laryngeal nerve as well as pilocarpine activated the secretion from the tracheal glands while atropine inhibited this action The goblet cells however, did not seem to be influenced by nervous stimulation

Lucas & Douglas (1935) in their ciliary beat studies on turtle trachea *in vivo* attributed the variations in mucus flow brought about by injection of *i a* pilocarpine, atropine and morphine to quantitative and qualitative changes in the mucous layer and not to direct action on the beat rate

Herrmann (1934) observed by means of X rays, the transportation of radio opaque particles in the bronchial tree of dogs and stressed the importance of an intact mucosa. In front of artificially created deciliated areas the mucus stopped for a long time

Although the previous investigations were made on living animals physiological conditions were not observed and thus the results were of limited value Variations in the temperature and relative humidity of the air passing the ciliary mucosa are known to influence the beat rate and consequently probably also the flow rate

Dalhamn (1956) made an extensive study of the ciliary beat rate and the speed of mucus flow in the rat trachea under normal as well as pathological conditions using a specially designed chamber where temperature and humidity could be controlled The average speed of mucus flow in a normal series was found to be 13 5 mm per minute when the air was saturated with humidity and had a temperature of 31°C In comparative *in vitro* and *in vivo* studies it was found that mucus transportation ceased within a relatively short time after the trachea was extirpated A reduction of relative humidity brought about a quick cessation of ciliary activity Elevation of the tissue temperature caused acceleration of the flow rate up to a temperature of 39 C but the beat rate showed no significant change Reduction of both the chamber and the rectal temperature to 25 2°C and 31 2°C respectively resulted in a significant decrease of the flow rate The technique was further developed in 1960 in such a way as to permit observation of the tracheal mucosa with the microscope in air tight connection with the tracheostomy wound enabling the animal to use the normal upper airways for breathing (Dalhamn 1960)

Krueger & Smith (1958a, b) and Krueger *et al* (1959) used a similar chamber for their *in vivo* experiment on the effects of negative and positive ions on mouse rat and monkey trachea at 25°—30 C chamber temperature and a relative humidity of more than 90 per cent The progress of air bubbles or grains of talc within a given time was measured by means of an ocular micrometer in a 30—45 power microscope The

normal mucus flow rate was found to vary widely, even among individuals of the same species The authors confirmed the findings of Dalhamn (1956) that the mucus flow rate remained relatively constant for the first few hours and then declined gradually, but without actually coming to a stop Dalhamn found transportation to be unaltered 80 minutes after the trachea was opened, but after 120 minutes it had been affected Ciliary activity, as observed from the beat rate, showed no change even after 120 minutes The absolute flow rates in the investigation of Krueger & Smith (1958a) are, however, much lower than those found by Dalhamn and varied between 2 mm and 6 mm per minute in the beginning of the observation period, before any ions had been administered From their results, Krueger & Smith drew the conclusion that the secretion of mucus and the actual rate of mucus flow are less susceptible to the action of positive ions in living animals than they are in isolated tracheal strips

The conclusion from the previously discussed animal studies of the mucus flow rate must be that *in vitro* studies are of a limited value and should not be used for investigations of the rate of flow Changes in temperature and relative humidity have been shown to influence the velocity of the mucus flow and should therefore be avoided, but even under constant conditions mucus flow decreases after a certain time, when the trachea is opened, and the animal remains under the influence of anaesthetics Strict physiological conditions, preferably without surgery and drug administration, must thus be aimed at

## Human studies

*In vitro* Although numerous reports concerning *in vitro* studies of the flow rate in human ciliary mucosa have appeared, the subject will not be dicussed, as the same limitations exist as those already mentioned in the previous section in connection with animal *in vitro* studies

*In vivo* With the basic technique used by Sharpey in the 1830s Yates (1924) and Hilding (1931a) mapped the ciliary pathways of the human nose and nasopharynx Yates did not stress the importance of his findings, but his study, being the first of its kind on living humans, stimulated many other authors to undertake ciliary research Both Yates and Hilding used a coloured substance and noted the path taken by it across the nasal and pharyngeal mucosa by direct observation, they found that the pathways were invariably directed towards the pharynx Hilding also measured the speed in different parts of the nose, finding the highest values in areas protected from the blasts of the inspired air The average speed was 4—6 mm per minute on the lateral wall and the fastest rate found was 10 mm per minute, observed on the septal mucosa of one person

Frenckner (1939) observed the movements of small pieces of paper across the mucosa of the nose and accessory sinuses of humans before and after radiation with X ray or radium tubes He considered that radiation decreased or arrested ciliary motility and mucus flow, at least temporarily The speed observed in five non radiated normal cases, however, was approximately 5 mm per minute, a value in accordance with more recent studies

Ornston (1946) used as tracers sulfathiazole powder, sometimes in heavy amounts and observed the pathways in the nose of his patients No remarks were made on the flow rate, which was observed with a nasopharyngoscope and posterior mirror examination

Tremble (1948) used a similar technique but measured the time taken for the indicator substance to appear in the nasopharynx as observed with a mirror By making a rough estimate of the distance travelled the speed was evaluated and found to be approximately 5 mm per minute in the normal cases Local anaesthetics and vasoconstrictors were sometimes used rendering the results of limited value

Using the same technique but without local anaesthesia and decongestant agents, van Ree & van Dishoeck (1962) found the average speed across the posterior two thirds of the nose in 106 cases to be 5 mm per minute with 3 mm and 8 mm per minute as extremes The investigation was carried out at room temperature over a period of one year, but nothing is mentioned regarding the relative humidity of the air The inexact measuring of the distance travelled renders the method used by these authors less suitable for scientific purposes but it has one great advantage consisting in the maintenance of physiological conditions

## *Conclusions*

Previous investigations of the ciliary activity and its resultant the mucus flow, have shown that *in vitro* studies should only be used for observations of the ciliary beat rate

One of the prerequisites for a normal mucus flow is an intact blood and nerve supply to the mucosa This means that *in vivo* studies are essential where mucus flow rate is concerned

*In vivo* investigations regarding the mucus flow rate in humans are very few in number and those already published have not been made under controlled and physiological conditions nor is a technique yet available for studies under such circumstances

In order to advance any further in this respect it is thus necessary to find an exact method for studying the mucus flow rate in humans under physiological conditions

# Method

## *Introduction*

Mucus flow is the result of many factors, and during optimal flow all these factors can be assumed to co-operate under optimal conditions The main factors can be summarized as follows

| | |
|---|---|
| *Mucous factor* | *Ciliary factor* |
| Amount of secretions | Number of cilia |
| Viscosity of secretions | Ciliary beat rate |

*Mucous factor* The term secretions' refers to the fluid layer covering the mucosa regardless of origin exudate or transudate or combination of both The question of origin is still open to discussion (Florey *et al*, 1932 Ingelstedt & Ivstam, 1949a, b Messerklinger, 1951 Negus 1958 1963)

The *amount of secretions* is an important factor regarding the speed of flow According to Gray (1928) the effect of ciliary activity on particles placed at a distance *of more than four or five times the length of the cilia becomes very small* This has also been demonstrated by various authors (Lommel, 1908 Hill 1928 Tremble 1948 Dalhamn 1956) It is a well known clinical fact that mechanical irritation *e g* touching of the mucosa causes an increase in secretion Florey *et al* (1932) corroborated this finding in animal experiments Consequently every effort should be made to work under physiological conditions without touching the mucosa

All attempts to find an easy and dependable method of measuring the *viscosity of the secretions* have failed This failure is probably due partly to the double layer nature of the mucus with a viscous surface layer on top of the watery deeper layer (Lucas & Douglas 1934) and partly to the relatively small amounts of secretions available under normal conditions (Dalhamn 1956 Toremalm 1960b) Negus (1958) points out that the pH value of mucus is about 7 0 *in situ* but about 7 4 when removed from the body for a while the higher value being due to loss of carbon dioxide Breuninger s (1964) already cited work on the gel and sol state of the nasal mucus at different pH levels around 7 5 to 7 6 should be mentioned in this connection

If owing to a decrease in the amount of secretions the viscous layer gets in contact with the cilia during the latter s recovery phase the forward movement of the layer will be hampered If on the other hand the viscosity is too low, the upward movement in the lower airways is impeded by gravity secretions come to a standstill or may even run back and accumulate in the finer bronchi leading to obstruction and atelectasis

*Ciliary factor* It can easily be understood that the *number of cilia* per unit area is important Each ciliated cell is considered to have 250—300 cilia (Rhodin, 1959) and as the ciliated cells are normally in a majority, the mucus layer is moved by an almost continuous carpet of cilia After local trauma however, with deciliated areas as after cautery, surgery, careless handling of instruments, etc, secretions pile up in front of these areas and are only slowly drawn by remote traction from active areas further back (Herrmann 1924, Hill, 1928, Hilding 1933 1957, 1959a) In an acute viral infection the surface epithelium is partly destroyed and shed ciliary fragments, still actively beating float in the secretions (Hilding 1959b) Before regeneration takes place, the mucus flow rate is naturally decreased over such areas

As has been discussed earlier *ciliary beat rate* is influenced by many factors, but the underlying mechanism remains unknown Under normal conditions cilia are seen to beat at a fairly constant rate of about 1000 beats per minute in animal *in vivo* studies (Dalhamn, 1956, Krueger & Smith 1958a, b Krueger *et al* 1959) A reduction of the beat rate results in a decrease of the flow rate (Dalhamn 1956) From *in vitro* studies on human ciliary mucosa, the average beat rate seems to be around 1000 per minute (Dalhamn *et al*, 1959, Krueger *et al*, 1959) but owing to technical difficulties no *in vivo* studies have yet been carried out on humans

*Conclusions* It is quite evident from the previous discussion that human *in vivo* studies of the involved factors taken separately would meet with great technical difficulties From a practical standpoint, however, the mucus flow rate under physiological conditions and its normal variations would be an important point of reference for further studies, especially if a technique could be employed that permitted repeated observations of the same area under controlled and variable conditions

## *Principles*

The nasal ciliated mucosa is the only place in humans that is directly accessible to observation of the mucus flow under physiological conditions It normally gets in contact with inspired air of varying temperature, humidity and grade of pollution with foreign materials, such as dust, soot bacteria and irritant gases, this provides possibilities of using similar agents in our experiments without disturbing the normal environment of the mucosa On the anterior part of the middle third of the septum the mucus flow usually goes in a dorsocaudal direction (Hilding 1931a van Ree & van Dishoeck 1962) enabling observation at right angles to be made (Fig 2)

The present method of observing the mucus flow is based on the following principles

1 Direct observation of the mucus flow under magnification
2 Visualization of the mucus flow with a non irritating indicator
3 Exact measurement of a certain distance and the time taken for the indicator to travel that distance
4 Maintenance of physiological conditions without premedication local or general anaesthesia or direct contact with the mucosa

*Ad 1* A Zeiss Diploscope was used in the present investigation It consists of two binocular operating microscopes mechanically connected with each other and with a

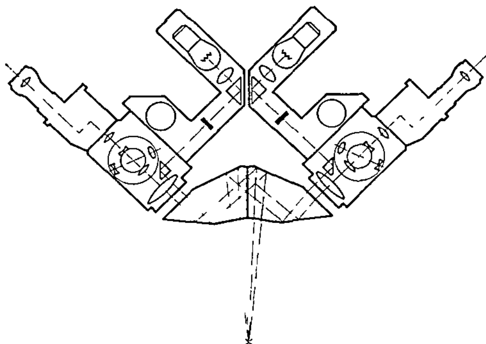


Fig 1 Diagrammatic sketch of the Diploscope

prismatic system that unites the illuminating and observation ray paths of both microscopes symmetrically in a central axis (Fig 1) The Diploscope can easily be brought into different positions and has a working distance of 230 mm (objective f=400 mm) When fitted with 20 × oculars five different magnifications are available 3, 5, 8, 12 5 and 20 × According to the manufacturers the fields of view with these magnifications are 62, 40, 25, 16 and 10 mm respectively Each microscope has a 6 V 50 W bulb as illumination source and is fitted with a built in heat filter

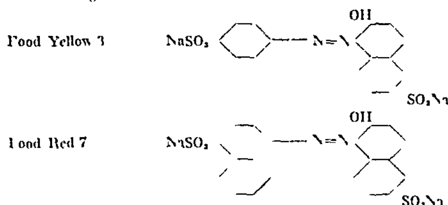
The advantage of a Diploscope over an operating microscope lies in the possibility of equipping the microscope, not used by the observer, with a camera Excellent illumination and a stereoscopic view of the observation area is thus easily obtained together with photographic recording facilities

*Ad 2* To visualize the otherwise invisible flow of the thin, transparent mucus layer an indicator is necessary The use of artificial tracer substances has been deprecated (Dalhamn, 1956 *i a* ), on the grounds that different particles may move at different speeds This is probably true when clogging of the particles takes place, causing localized drying and change in viscosity, especially when observed under high magnification and over a short distance The main objection to the use of a tracer substance, however, lies in the fact that the particles must be assumed to move at the same speed as the mucus This is probably true as long as they are not too big and as long as they adhere to the viscous surface layer, thereby enabling the clearance ability of the mucosa to be measured

It is possible that the two layers move at different speeds A highly viscous surface layer may be stagnant, as would any particle embedded in it, while a small particle slightly below may be moved by the tips of the beating cilia On the other hand, a particle that is close to the epithelial surface may be trapped by the cilia, while the surface layer is moving This could explain the finding of Dalhamn (1956) with particles of equal size, moving at different speeds

Owing to the surface tension of the superficial layer, freshly applied powder particles can be assumed to remain on the surface and indicate the flow of the layer during the short period of observation It is also the experience from the present investigation that the majority of particles travel with the same velocity once they arrive at an active area

The indicator substance originally used by Tremble (1948) and recently by van Ree & van Dishoeck (1962) was a chemically inert powder, composed of dibasic calcium phosphate, 97 per cent, and coloured by a foodstuff dye Edicol Orange, 3 per cent The colour substance, used in the same proportion in the present study, is manufactured by Imperial Chemical Industries Ltd, England under the name of Edicol Supra Orange AG and consists of Edicol Supra Yellow FCS (C I Food Yellow 3, No 15985) and Edicol Supra Ponceau 4 RS (C I Food Red 7, No 16255) which have the following chemical formula




When dry the indicator powder was greyish pink without taste or smell but when moist it was bright orange and easily visible although the particles observed were less than 0 1 mm in diameter They were situated high on the mucous layer and did not clog The powder was used throughout the present investigation without causing any noticeable side-effects such as local irritation discomfort allergy or drying It is however important to apply only a few particles at a time otherwise artefacts like those mentioned above may occur

For this purpose a small powder blower was used and was found easy to handle, without touching the mucosa

*Ad 3* The distance travelled by the indicator particles across the observed area was measured without touching the mucosa by fitting one ocular piece of the microscope with a calibrated scale adjusted in the plane through the optical centres of the two

oculars The time taken for the particles to travel a certain distance, 2 mm, was measured with an ordinary stop watch, marked in tenths of a second Even though the microscope was without apparent spherical aberration, only the central fifth of the field of view was used for measuring

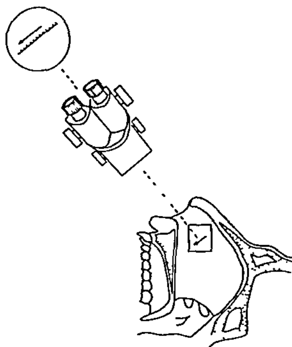


Fig 2 The correct position of the Diploscope for obtaining a perpendicular view of the observation area and the mucus flow (arrow)

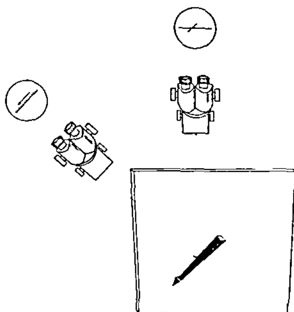


Fig 3 Schematic view of the square area in Fig 2 The correct position of the Diploscope yields an ocular image where the flow line and the scale are parallel while in the incorrect position they are intersecting

Scale measurement technique presupposes observation at right angles To attain this the following criteria must be fulfilled

1, The scale and the line of flow must be parallel
2 The total area along the line of flow must be in focus, including the extremes even with the highest magnification

The importance of a strict adherence to these criteria can be explained by the following reasoning

Fig 2 shows a schematic view of the septum the area used for observation in this study, and the approximate direction of flow of mucus in this area The correct line of observation perpendicular to the line of flow is also marked as a dotted line extending through the nostril At the outer end of this line is the view as seen in the microscope with the line of flow (arrow) and the scale running parallel

The square area in Fig 2 is shown in Fig 3 The arrow marks the flow, going in a dorsocaudal direction When an incorrect line of vision not going perpendicularly to the line of flow, is used the arrow and the scale, fixed in the plane through the optical centres of the oculars are not parallel with each other

The septum and subsequently the line of mucus flow sometimes go in an oblique plane as in septal deviations That is why the microscope must be adjusted to the same plane by bringing the two extremes of the field of view along the line of flow (and the scale) into focus By observing criterion No 2 a perpendicular line of view is secured

Using the highest magnification 20 ×, the maximum depth of focus will be 2 mm which means a source of error in the measuring technique

The extent of this error can be determined through the following reasoning

In Fig 4 the distance AB marks the diameter of the field of vision with 20 × magnification 10 mm and the distance $BB_1$ the maximum depth of focus 2 mm The points A and $B_1$ are thus within focus and the distance $AB_1$ is the maximum distance allowed with observance of criterion No 2 The angle $\alpha$ between the lines AB and $AB_1$ will be the angular error under such circumstances

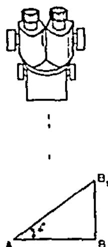



Fig 4 AB Diameter of the field of view at 20 × magnification BB Depth of focus at 20 × magnification The hatched area is out of focus

Using the numerical values for the distances AB and $BB_1$ in the formula $(AB)^2 + (BB_1)^2 = (AB_1)^2$ yields $AB_1 = 10.2$ The angle $\alpha$ is obtained in the same manner from the formula $tg\ \alpha = \frac{BB_1}{AB}$ $\alpha = 11.3°$

Consequently the methodical error associated with the distance measuring technique is less than 3 per cent, or less than 0.1 mm as the distance observed in this study is 2 mm, with a tolerated angular error of approximately 11° from the perpendicular plane

In order to estimate the error from a practical standpoint, a piece of cardboard was fixed in a vertical position, simulating the septum The Diploscope was focused on a certain area, following the criteria, and a distance of what was considered 2 mm was marked off with a fine-pointed pencil, using the scale for measuring at 5 × magnification This procedure was repeated 100 times The distances marked were then measured with 20 × magnification at a controlled 90° angle The mean value was 2.01 mm with a standard deviation of 0.05 mm The practical evaluation was thus in good accordance with the theoretical analysis

The times read from the stop watch were approximated to whole seconds when recorded, the maximum error thus obtained being 0.5 seconds or 5 per cent, as the shortest times recorded were 9—10 seconds Two or more readings were made in rapid succession and their mean value was used for calculating the speed, thereby lessening this source of error

*Ad 4* To test the built in heat filters of the Diploscope, a standard mercury laboratory thermometer graded in tenths of degrees C was placed at room temperature under maximal illumination for 30 minutes, without changing more than 0.5°C from the original value The short observation times during the present study, about 2 minutes the effective heat regulating mechanism of the nasal mucosa and the constant streaming of air over the illuminated area serve to minimize this source of error

By placing the subject comfortably on his back on an operating table and gently widening the nares with a nasal speculum only touching the vestibular epithelium and by carefully applying only a few powder particles at a time with the blower, without touching the mucosa, discomfort to the subject is avoided No premedication or anaesthesia is needed and the normal physiology of the nose is not disturbed, with one exception By widening the nares, which is necessary in almost all cases, the inspired non-conditioned air is caused to follow a route that is different from the normal one, that curves upwards toward the roof (Proetz, 1953) Leading the air straight backwards may possibly influence the mucosa causing neural and vascular reactions If the observation times are kept as short as possible these artefacts will, however, be negligible

The influence of gravity was tested in a small group by comparing the speed of flow with the septum in the vertical and horizontal positions without finding any differences This is in good accordance with earlier investigations (Hilding, 1931b, Negus 1958 van Ree & van Dishoeck, 1962) where gravity was shown to be of no importance in animal and human *in vitro* and *in vivo* studies The effect of gravity under

Scale measurement technique presupposes observation at right angles To attain this the following criteria must be fulfilled

1 The scale and the line of flow must be parallel
2 The total area along the line of flow must be in focus, including the extremes, even with the highest magnification

The importance of a strict adherence to these criteria can be explained by the following reasoning

Fig 2 shows a schematic view of the septum, the area, used for observation in this study, and the approximate direction of flow of mucus in this area The correct line of observation, perpendicular to the line of flow, is also marked as a dotted line extending through the nostril At the outer end of this line is the view as seen in the microscope with the line of flow (arrow) and the scale running parallel

The square area in Fig 2 is shown in Fig 3 The arrow marks the flow, going in a dorsocaudal direction. When an incorrect line of vision, not going perpendicularly to the line of flow, is used the arrow and the scale, fixed in the plane through the optical centres of the oculars, are not parallel with each other

The septum, and subsequently the line of mucus flow, sometimes go in an oblique plane as in septal deviations That is why the microscope must be adjusted to the same plane by bringing the two extremes of the field of view along the line of flow (and the scale) into focus By observing criterion No 2, a perpendicular line of view is secured.

Using the highest magnification, 20 ×, the maximum depth of focus will be 2 mm, which means a source of error in the measuring technique

The extent of this error can be determined through the following reasoning

In Fig 4 the distance AB marks the diameter of the field of vision with 20 × magnification, 10 mm, and the distance $BB_1$ the maximum depth of focus, 2 mm. The points A and $B_1$ are thus within focus and the distance $AB_1$ is the maximum distance allowed with observance of criterion No 2 The angle $\alpha$ between the lines AB and $AB_1$ will be the angular error under such circumstances

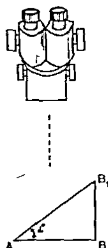



Fig 4 AB Diameter of the field of view at 20 × magnification $BB_1$ Depth of focus at 20 × magnification. The hatched area is out of focus

the table, is fixed in a position above the head of the subject, whose nose is in the midline The observer, sitting at the left side of the table, brings the Diploscope to focus on the septum, by tilting it in the sagittal plane and by gently widening the nares with a nasal speculum held in his left hand A slightly oblique line of vision (10°—20°) from the septal plane gives the best surveillance, using the 5 × magnification (Fig 5)

A few particles are now blown on the septal mucosa, approximately 3 cm from the orifice, to test for mucus flow and to make the final adjustments of the tilting angle according to the two criteria mentioned above This is done under 20 × magnification for the reasons discussed earlier The 5 × magnification is then switched back into position, and the time taken for the powder particle to move a distance of 2 mm along the calibrated scale is recorded An area with uniform mucus flow should always be sought for and is usually easily found

It is necessary to have a point of reference on the mucosa to coincide with a certain point of the scale The small vessels, clearly visible through the transparent mucous layer at 5 × magnification, are ideal for this purpose

By turning the Diploscope in the frontal plane, the same procedure is repeated on the opposite side of the septum At least two readings are done on each side and the speed of flow is computed from their mean value, using the formula

$$\text{Speed in mm/min} = \frac{2 \times 60}{\text{Time in seconds}}$$

The distance used, 2 mm, was found to be appropriate, the average time of flow along it being 30 seconds A shorter distance would increase the errors and a longer one would take too much time to measure and for the subject to lie still In those few cases where the flow was extremely slow and the times were longer than 120 seconds for 2 mm, the speed was marked as '0', as were also the cases where no movement could be noticed during 2 minutes on comparing the particles with the reference points at regular intervals

# Material

In the following chapter the results from a study of a material, using the previously described technique will be analysed It should be observed that all data are obtained from a very limited area on the septal mucosa, the results must therefore not *a priori* be considered as relevant to other parts of the respiratory system, or even to other parts of the nose The area was deliberately chosen, as being the only place with ciliated mucosa, normally exposed to intermittent blasts of non conditioned air and accessible to observation with the present technique Septal deviations and cyclic variations in the volume of the mucosa of the turbinates (Heetderks, 1927, Beickert, 1951, Stoksted, 1952) change the direction of the air stream, and consequently the very same area will sometimes be exposed to non conditioned air and at other times sheltered Relatively large intra-individual variations between the two sides of the septum, as well as inter individual ones, may thus be expected

The purpose of the investigation was to study the mucus flow rate (FR) in the human nose and the effect of the following factors on this rate

The relative humidity (RH) of the ambient room air

The smoking habits and the age and sex of the subjects

The evolved technique has been used in a number of cases which could be divided into two main groups

1 Subjects breathing through their upper respiratory tract

2 Subjects breathing through a tracheostomy

The normally breathing subjects in group 1 are called the ***normal group*** as they can be regarded as a sample (although not in a statistical sense) of the normal population with men and women of different age and smoking habits The tracheostomized group 2 will be referred to as the ***tracheostomy group***

The large normal group has been used for a statistical analysis regarding the mucus flow rate, while the tracheostomy group, being much smaller and not suited for such an analysis, was used to elucidate some findings, obtained from the study of the normal group

*The normal material* is based on data obtained from 174 examinations, all of them done bilaterally on the septum, and carried out on 165 subjects, a few of whom were examined more than once, but under different humidity conditions

The subjects were either patients at the Ear, Nose and Throat Clinic, Karolinska Sjukhuset, Stockholm, who had been hospitalized for other ailments than those of the respiratory tract, *(e g* otosclerosis, tumours of the salivary glands, oesophageal diverticula) or members of the hospital staff All were interrogated and examined by the author, and only those with normal rhinoscopic findings were accepted A slight septal deviation was tolerated as long as it did not cause any subjective or objective

signs of discomfort, such as dry membranes, crusts, frequent infections or blocking of the passage Cases with allergy, vasomotor rhinitis, acute respiratory tract infection and those giving a history of previous surgery or radiotherapy of the nose or nasal sinuses were not included The rectal temperatures of all subjects were less than 37 5°C on the day of examination and none were receiving medication known to have any influence on the nasal mucosa

*The tracheostomy group* is made up by 25 subjects, 12 of which had been tracheostomized or laryngectomized for more than a month, while the others were studied on different occasions during the first postoperative month in order to observe the effects on the nasal mucosa, produced by the operation

The complete basic data for the normal group and the 12 cases, tracheostomized for more than a month are given in Tables 1, 7 and 9—17 Only Tables 1 and 7 are placed in the text section whole the other tables appear separately on pp 56—62 for the sake of clarity

Heavy smokers smoked >10 cigarettes or >2 cigars a day or >50 g pipe tobacco a week Moderate smokers smoked <10 cigarettes or <2 cigars a day or <50 g pipe tobacco a week and the non smokers had not been smoking for 3 months

*Table 1 Normal group Relative humidity less than 40 per cent Age 30 years and younger N Non smokers M moderate smokers H heavy smokers RH Rel humidity FR Flow rate*

| Case | RH | Age | Sex | Smoking habits | Fast side: Time s/2 mm: Observation I | II | III | Mean | FR mm/min | Slow side: Time s/2 mm: Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 159 | 26 | 22 | ♀ | N | 20 | 21 | 22 | 21 | 5 7 | 25 | 25 | | 25 | 4 8 | 5 2 |
| 137 | 28 | 27 | ♂ | M | 50 | 55 | 52 | 52 | 2 3 | ∞ | | | ∞ | 0 0 | 1 2 |
| 142 | 29 | 19 | ♂ | M | ∞ | | | ∞ | 0 0 | ∞ | | | ∞ | 0 0 | 0 0 |
| 83 | 29 | 27 | ♂ | N | 15 | 15 | 15 | 15 | 8 0 | 17 | 17 | 20 | 18 | 6 7 | 7 4 |
| 133 | 30 | 20 | ♀ | M | ∞ | | | ∞ | 0 0 | ∞ | | | ∞ | 0 0 | 0 0 |
| 116 | 30 | 23 | ♂ | M | ∞ | | | ∞ | 0 0 | ∞ | | | ∞ | 0 0 | 0 0 |
| 8 | 30 | 26 | ♀ | N | 20 | 20 | 20 | 20 | 6 0 | ∞ | | | ∞ | 0 0 | 3 0 |
| 84 | 32 | 23 | | M | 27 | 27 | 27 | 27 | 4 4 | ∞ | | | ∞ | 0 0 | 2 2 |
| 78 | 33 | 12 | | N | 11 | 11 | 11 | 11 | 10 9 | 17 | 15 | 15 | 16 | 7 5 | 9 2 |
| 77 | 33 | 24 | | N | 12 | 12 | 12 | 12 | 10 0 | 15 | 15 | 15 | 15 | 8.0 | 9 0 |
| 7B | 33 | 25 | | N | 30 | 30 | 30 | 30 | 4 0 | 35 | 35 | 35 | 35 | 3 4 | 3 7 |
| 69 | 33 | 25 | | N | 20 | 20 | 20 | 20 | 6 0 | ∞ | | | ∞ | 0 0 | 3 0 |
| 89 | 33 | 27 | ♀ | N | 11 | 11 | 11 | 11 | 10 9 | 30 | 30 | 30 | 30 | 4 0 | 7 4 |
| 120 | 33 | 27 | | N | 14 | 14 | 14 | 14 | 8 6 | 14 | 14 | 14 | 14 | 8 6 | 8 6 |
| 95 | 33 | 30 | ♂ | H | 20 | 20 | 20 | 20 | 6 0 | 25 | 25 | 25 | 25 | 4 8 | 5 4 |
| 104 | 34 | 14 | ♀ | M | 40 | 40 | 40 | 40 | 3 0 | 43 | 43 | 40 | 42 | 2 9 | 3 0 |
| 115 | 34 | 23 | ♂ | M | ∞ | | | ∞ | 0 0 | ∞ | | | ∞ | 0 0 | 0 0 |
| 166 | 35 | 23 | ♂ | H | 22 | 22 | 22 | 22 | 5 5 | ∞ | | | ∞ | 0 0 | 2 8 |
| 67B | 35 | 25 | ♀ | H | 15 | 15 | 15 | 15 | 8 0 | 20 | 20 | 20 | 20 | 6 0 | 7 0 |

# Material

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## *Normal group*

### Statistical analysis

The statistical analysis was carried out with product moment correlation The significance of the coefficients of correlation were tested in the formula

$$t = r \sqrt{\frac{(n-2)}{(1-r^2)}}$$

r Coeff of correlation
n Number of cases

The coefficients of correlation for RH *vs* FR and Age *vs* FR of the different sex and smoking groups are given in Table 4

*Table 4 Correlation between mucus flow rate, FR, and relative humidity, RH, and age*

| Smoking habits | n | Coefficient of correlation RH/FR | Age/FR |
|---|---|---|---|
| Male moderate | 26 | 0 77 | 0 33 |
| Male heavy | 20 | 0 45 | 0 03 |
| Male non smokers | 39 | 0 26 | 0 11 |
| Female moderate | 17 | 0 71 | —0 17 |
| Female heavy | 14 | 0 60 | 0 05 |
| Female non smokers | 58 | 0.26 | 0 19 |

The groups *male moderate* and *heavy smokers* and *female moderate* and *heavy smokers* are combined to *male smokers* and *female smokers* respectively in the following because of their similar patterns and the fact that only a very few "heavy smokers smoked more than one pack of cigarettes a day

The results of the correlation analysis for the factors RH *vs* FR, and Age *vs* FR are given in Table 5

Correlation is thus seen to exist between relative humidity and mucus flow rate, but there is no significant correlation between age and mucus flow rate ($P > 0\,05$) The correlation is statistically significant for smokers of both sexes at a level of $P < < 0\,001$ and for female non smokers at a level of P 0 05—0 01 A slight enlargement of the male non smoking group would probably have yielded a significant correlation

The time taken by the particle to travel 2 mm was recorded from 3 observations and is marked under the heading "Time s/2mm, Observation I, II and III (In a small number of cases only 2 observations were made, owing to technical difficulties) The mean time value is marked in a special column and the FR determined from this mean The headings "Fast side' and "Slow side" have been used because in the majority of cases there is a difference between the two sides of the septum

The mean flow rate values from both sides are listed in the last column and have been used in the following statistical analysis for the following reason As large intraindividual variations can be expected due to reasons, already mentioned, the mean flow rate of the two sides is used because of the equalizing effect of such a procedure

Relatively dry inspiratory air, alternating with over saturated expiratory air pass in a turbulent stream across the septal area used for observation (Ingelstedt & Toremalm, 1961) Owing to technical and physiological reasons exact measurements of the relative humidity of this turbulent air is not feasible It can, however, be reasonably assumed that the relative humidity of the air over the observation area is not lower than that of the ambient air and, consequently, any found correlation between flow rate and relative humidity will appear to be less significant than it actually is by using the ambient relative humidity value instead of the actual higher value at the area of observation

The composition of the normal group is given in Tables 2 and 3

*Table 2 Normal group Number of cases grouped according to smoking habits*

| Smoking habits | Male | Female | Total |
|---|---|---|---|
| Smokers | 46 | 31 | 77 |
| Moderate smokers | (26) | (17) | (43) |
| Heavy smokers | (20) | (14) | (34) |
| Non-smokers | 39 | 58 | 97 |
| Total | 85 | 89 | 174 |

*Table 3 Normal group Number of cases grouped according to the relative humidities at the time of examination and the age of the subjects RH Rel humidity*

| | Age groups | | | |
|---|---|---|---|---|
| RH groups | <31 years | 31—60 years | >60 years | Total |
| <40 % | 19 | 46 | 13 | 78 |
| 40—59 % | 21 | 28 | 10 | 59 |
| 60 79 % | 10 | 13 | 8 | 31 |
| 80 % and above | 3 | 3 | 0 | 6 |
| Total | 53 | 90 | 31 | 174 |

*Table 6 Statistical analysis RH Rel humidity*

| Group | n | Relative humidity/Flow rate | | | Flow rate | |
|---|---|---|---|---|---|---|
| | | Coeff of correlation | P | Coeff of regression | Unstandard mean/RH | Standard mean/RH |
| Smokers | 77 | 0 674±0 062 | <0 001 | 0 107±0 014 | 3 47/42 8 | 3 55/43 55 |
| Non-smokers | 97 | 0 273±0 094 | 0 01—0 001 | 0 047±0 017 | 4 80/44 3 | 4 76/43 55 |
| Total normal group | 174 | 0 435±0 062 | <0 001 | 0 075±0 012 | 4 22/43 6 | |

between the unstandardized and the standardized means (P<0 001) (Analysis of covariance) This implies that the smokers have a lower flow rate average than the non smokers At higher relative humidity levels, however, this condition seems to be compensated for

In Fig 7 the total normal group is finally plotted A few cases with identical values for RH and FR are only marked once As can be seen the majority of cases lie between RH 30 and 60 per cent, but the line of regression is nevertheless continued to RH 0 and 100 per cent respectively

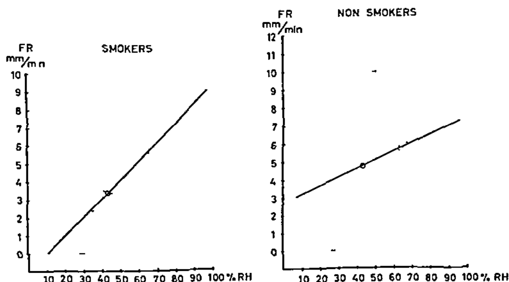



Fig 7 Cases with identical values are plotted only once The circles mark the standardized means at 43 55 per cent relative humidity RH FR Flow rate

## Normal mucus flow rate

Earlier studies on humans (Hilding, 1931a, Frenckner, 1939, Tremble, 1948, van Ree & van Dishoeck, 1962) give an average mucus flow rate through the nose of approximately 5 mm per minute As earlier stated, these reports do not mention any data on temperature and relative humidity or the age and sex of the subjects, the methods are less exact and sometimes unphysiological

*Present results* As can be seen in the statistical analysis, no absolute, normal mucus flow rate can be determined, as the rate is subjected to wide variations, due to different factors, such as the relative humidity of the inspired air The mean flow rate in the observed area of the normal group was, however, 4 2 mm per minute at 43 6 per cent RH

## Relative humidity

The nose plays an important role in the conditioning of the inspired air (Heetderks, 1927, Perwitzschky, 1928, 1930, Seeley, 1940, Ingelstedt & Ivstam, 1951, Cole, 1953a, b, 1954a, b, Ingelstedt & Toremalm, 1960, Toremalm, 1960a) Ingelstedt (1956) could show that the inspired air during nasal breathing was almost fully saturated with humidity and warmed to more than 30°C, when passing the subglottic region, regardless of whether the ambient air was temperate and dry or cold and moist

A total of approximately 560 g of water is needed to saturate the normal daily respiratory volume of 15 m³ of ordinary room temperature (21°C) and RH 35 per cent The major part is derived from the upper respiratory tract mucosa, and the nose is supposed to produce approximately 430 g a day, 130 g of which, however, has been condensed on the relatively cool membranes during expiration (Toremalm 1960a), leaving a total rest of approximately 300 g a day to be secreted by the nasal mucosa

The area, observed with the present technique is situated at the beginning of the nasal passage, permitting us to assume that the air, streaming over that region, is relatively non conditioned during the inspiratory phase During expiration, however, the air is oversaturated and an amount of water is condensed on the mucosa

The efficiency of the conditioning mechanism of the nose is partly due to the close contact between the streaming air and the mucosa, caused by the turbulent air flow, known to exist in the human nose (Ingelstedt & Toremalm, 1961) Turbulence is also known to exist in the subglottic region owing to the constrictor action of the vocal cord region During forced respiration this turbulent area may continue into the main bronchi

The aerodynamical conditions of the nose and the trachea are thus in many respects similar to each other, making the study of the ciliary function of the nose important, not only for its own cause but for a better understanding of the mechanism of bronchial pathology Non conditioned and polluted air may under certain circumstances reach the lower respiratory tract and damage the respiratory epithelium, causing stagnation of secretions From the experiments of *i a* Dalhamn (1956) and Toremalm (1961) cilia are known to cease beating within 30 minutes of exposition to air

of less than 50 per cent relative humid[illegible] humidity to 70—80 per cent kept the [illegible] conditions

As dry air is known to cause such [illegible] would be of great interest to see if this [illegible] conditions

*Present results* The statistical analys[illegible] mucus flow rate and relative humidit[illegible] ±0 062, P<0 001) and non smokers [illegible] 0 001) as well as in the total normal [illegible] P<0 001) as seen in Table 6

The effect of dry air on the mucus flo[illegible] the basic data in Tables 1, 9—17 where [illegible] is relatively high when RH is less than [illegible] compared to the higher RH groups, w[illegible] side', however, FR 0 can be seen even [illegible] relative humidity, the greater the numb[illegible] mately 60 per cent below RH 40 per cent

*Comments* Once having established [illegible] tive humidity, studies of the flow rate a[illegible] of a definite interest Due to climatic con[illegible] relatively few, making the data obtained [illegible] statistical analysis The extremes of the [illegible] therefore be considered as uncertain and [illegible] straight one can not be excluded

The basic data can however, give some [illegible] humidity seems to be the approximate l[illegible] can be seen to stop the mucus flow in a[illegible] relative humidity gets Above the 70 per [illegible] present material, but the number of subje[illegible] small for definite conclusions

Below 40 per cent relative humidity the [illegible] sation is not sufficient for keeping the [illegible] greater the number of cases with FR 0 [illegible] per cent—60 per cent relative humidity a[illegible] with the present results

[illegible]

*Conclusions* In the normal group a h[illegible] to exist between relative humidity and [illegible] humidity no harmful drying effect is n[illegible] relative humidity the drying effect is p[illegible]

evaporation from the mucosa but below 40 per cent relative humidity the drying effect preponderates

## Smoking habits

The depressant effect of tobacco smoke on ciliary activity has been demonstrated by various authors Kreuger & Smith (1958b) found that cigarrette smoke promptly decreased ciliary activity in the living rabbit's trachea by about 200 beats per minute, but when the smoke was flushed out, the ciliary beat rate returned at once to its initial level Hilding (1956a, b), Falk *et al* (1959), Kotin & Falk (1960) and Kensler & Battista (1963) found cigarette smoke to interfere with the mucus flow during *in vitro* experiments Dalhamn (1959, 1964) and Ballenger (1960) have shown that cigarette smoke may, at least temporarily, decrease or stop ciliary beating *in vitro* as well as *in vivo* in animals Owing to the lack of a proper technique no human *in vivo* studies of tobacco smoke and ciliary activity or mucus flow rate have been published

Positive ions are present in cigarette smoke according to Public Health Service Publication No 1103 (1964) and such ions have been shown to augment and sustain the reduction in ciliary beat rate, occuring after exposure to cigarette smoke, during a prolonged time (Krueger & Smith, 1958b)

Morphologic alterations of the respiratory epithelium with diminished length of the cilia and reduction of ciliated cells as well as basal cell hyperplasia and presence of atypical cells have been demonstrated in smokers as compared to non smokers (Ide *et al*, 1959, Auerbach *et al*, 1962a, b)

The effect of cigarette smoke on the quantity and quality of mucus has not been studied, but the known sympathomimetic effect of nicotine, causing a peripheral vaso constriction may probably affect the mucous factor in the highly vascular nasal mucosa with its goblet cells and autonomically innervated glands

*Present results* The analysis of the normal group (77 smokers and 97 non smokers) demonstrates a statistically significant difference between smokers and non smokers regarding the slope of the line of regression (relative humidity *vs* mucus flow rate) at a P level of 0 01—0 001 as well as the standardized means at 43 55 per cent relative humidity at a P level of $<0\ 001$ At lower relative humidity levels the difference in means seems to be even more marked but at higher levels it seems to be compensated for (Table 6 and Fig 7)

*Comments* As tobacco smoke is known to affect various factors involved in mucus transportation, disturbance of the flow rate in smokers would be expected

In the present material only the delayed effects of smoking can be studied as observations never took place immediately following smoking

When exhaling only a minor portion of the smoke usually passes through the nose after being inhaled and filtered by the lower respiratory tract The concentration and amount of harmful products in the expiratory air, passing the observed area can reasonably be assumed to be relatively small, when compared to the inhaled smoke charged portion A prolonged effect by substances, dissolved or entrapped in the

mucous layer can almost be disregarded in this particular area that is situated at the beginning of the mucus transportation route

The general effect of nicotine on the autonomic nervous system, however most probably affects the mucous factor for a prolonged time and could possibly cause such a decrease in flow rate, as the one seen in the present investigation, especially at lower relative humidity levels

*Conclusions* Statistical analysis of the normal group shows a significant difference in flow rate means between smokers and non-smokers ($P<0\,001$) Smokers have a lower flow rate mean, 3 6 mm per minute, than non smokers, 4 8 mm per minute at 43 55 per cent relative humidity At lower relative humidity levels the difference in means seems to be even more marked, while at higher levels the difference appears to diminish

The present study does not provide material for definite conclusions regarding the underlying mechanism, however, and the problem can only be solved by further investigations

## Age and sex

Old age is known to result in an atrophy of the mucosa with its glands (Wustrow, 1958) which in turn may lead to qualitative and quantitative changes in the secretions As human *in vivo* studies are very rare, age has not been correlated to ciliary activity until 1963, when Vasilenko found an impaired mucus flow in a group of 25 subjects, aged 75 to 102, compared to a group of 25 subjects 60 to 74 years old, by using a technique, similar to the one of Tremble's

The composition of the normal material regarding age is seen in Table 2

The mean age is 42 5 (12—80) years with more than 50 per cent of the cases in the middle-age group

Statistical analysis of this material does not show any correlation between age and mucus flow rate ($P>0\,05$), the highest mean 12 mm per minute is found in a subject, 66 years old

Sex has not previously been correlated to mucus flow rate in the respiratory tract Changes in ciliary activity in the Fallopian tubes, due to hormonal influence, have, however, been reported in rabbits (Borell *et al* 1957)

In the normal group, 85 men and 89 women, statistical analysis shows almost identical coefficients of regression and correlation for the male and female groups, regarding the correlation between relative humidity and mucus flow rate (Fig 6)

Intra individual variations during a period of time as during the menstrual cycle can naturally not be observed in the present material Repeated examinations of a relatively large number of women during different phases of the cycle would perhaps show variations in mucus flow rate, but the mean from single observations, as in the present study will only yield the mean of the total cycle

*Conclusions* No correlation could be demonstrated to exist between mucus flow rate and the age or sex of the subjects in the normal group

## *Tracheostomy group*

In the previous chapter the mucus flow rate in a control group has been studied under variable conditions Low relative humidity of the inspired air has been shown to diminish this rate and even to stop the flow completely below a certain level Wide variations have been demonstrated under similar environmental conditions, not only between different subjects but also between the two sides of the septum in the same individual These variations are assumed to be due to aerodynamic differences within the nasal cavities, bringing the mucosa in a more or less intimate contact with the non conditioned inspiratory air streams

Normal conditions are thus not identical with optimal conditions as regards the ciliary activity and its result, the mucus flow Only in the sinuses or the sheltered parts of the nose and the fine bronchi, where saturated air of a constant temperature slowly streams across the mucosa in a laminar flow, could almost ideal conditions be assumed to exist It would be of great interest to study the mucus flow under such circumstances, but the areas referred to are not accessible for observation with the present technique

The conditions in the nose of a tracheostomized patient are, however, very close to the ideal From earlier studies (Dixon *et al* , 1949, Schwab 1955) the nasal mucosa in tracheostomized patients is known to be well ciliated, with a rich supply of glandular tissue, always moist, with a good vascularization, a constant, relatively high temperature and minimal signs of inflammation

A group of tracheostomized patients were therefore made the object of a special study in order to elucidate the functional and morphological changes occurring in the nasal mucosa after a tracheostomy

### Functional changes

***Flow rate study*** Dixon *et al* (1949) and Schwab (1955) studied the mucus flow rate in the nose of tracheostomized patients, both of them using basically the same technique as Tremble (1948), by applying a coloured indicator to the anterior end of the inferior turbinate and the corresponding area of the septum and measuring the time taken for the indicator to appear in the nasopharynx The mean time varied between 9 5 minutes (Dixon *et al* ) and 7 4 minutes (Schwab) Schwab also measured the transportation time in a control grop and found the mean to be 9 minutes Neither of these authors gives any information on the relative humidities or the exact distances travelled by the powder and this makes the results unsuitable for comparison, but they both agree, that the flow rate seems to be faster in the tracheostomy group than in the normal one, and Schwab was able to correlate the speed to the higher mucosal temperature in the tracheostomy group, as compared to the control grop Within both groups the highest rates were found in the cases with the highest temperatures and the author suggests that changes in the secretions explain the difference

***Present material*** The basic data obtained from 12 subjects tracheostomized or laryngectomized for more than one month are given in Table 7 The group consists

*Table 7 Subjects tracheostomized more than one month*

| Case | RH % | Age | Sex | Postop time | Fast side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Slow side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 54 | 35 | 42 | ♂ | 7 ys | 13 | 13 | 13 | 13 | 9 2 | 17 | 17 | 17 | 17 | 7 1 | 8 2 |
| 66 | 39 | 62 | ♂ | 10 ys | 11 | 11 | 11 | 11 | 10 9 | 11 | 11 | 11 | 11 | 10 9 | 10 9 |
| 73 | 40 | 53 | ♀ | 9 ms | 12 | 12 | 12 | 12 | 10 0 | 12 | 12 | 12 | 12 | 10 0 | 10 0 |
| 76 | 33 | 52 | ♂ | 10 ys | 11 | 12 | 11 | 11 | 10 9 | 11 | 11 | 11 | 11 | 10 9 | 10 9 |
| 82 | 28 | 64 | ♀ | 7 ms | 9 | 9 | 9 | 9 | 13 3 | 9 | 9 | 9 | 9 | 13 3 | 13 3 |
| 150 | 16 | 71 | ♂ | 2 ys | 11 | 11 | 11 | 11 | 10 9 | 11 | 11 | 11 | 11 | 10 9 | 10 9 |
| 175 | 45 | 62 | ♂ | 10 ms | 15 | 15 | 15 | 15 | 8 0 | 15 | 15 | 15 | 15 | 8 0 | 8 0 |
| 197 | 39 | 75 | ♀ | 10 ys | 12 | 12 | 12 | 12 | 10 0 | 15 | 15 | 15 | 15 | 8 0 | 9 0 |
| 210 | 60 | 77 | ♂ | 14 ms | 12 | 12 | 12 | 12 | 10 0 | 12 | 12 | 12 | 12 | 10 0 | 10 0 |
| 219 | 40 | 36 | ♂ | 37 ds | 10 | 10 | 10 | 10 | 12 0 | 10 | 10 | 10 | 10 | 12.0 | 12 0 |
| 237 | 64 | 16 | ♂ | 4 ms | 11 | 11 | 11 | 11 | 10 9 | 11 | 11 | 11 | 11 | 10 9 | 10 9 |
| 239 | 42 | 58 | ♀ | 11 ms | 11 | 11 | 11 | 11 | 10 9 | 11 | 11 | 11 | 11 | 10 9 | 10 9 |
| Mean | 40 | 55 7 | | | | | | | | | | | | | 10 42 |

of 8 men and 4 women, mean age 55 7 years (range 16—77) and observed at varying relative humidities, mean 40 per cent (range 16—64) The mean flow rate obtained from the two sides of the septum is 10 4 mm per minute (range 8 0—13 3), the maximum intra-individual difference is 2 1 mm per minute, but 10 of the 12 cases show identical flow rates in both sides Although the group is to small for statistical analysis, flow rate and age, sex, relative humidity or postoperative time do not seem to be related The intra-individual and the inter-individual dispersion is also seen to be considerably less than in the normal group

*Comments* The relatively high and constant mucus flow rate found in the tracheostomy group favours the assumption that conditions are optimal The absence of evaporation keeps the water content of the mucus at a high level and lowers the viscosity The high and constant temperature, the stationary saturated air and the absence of irritation and infection contribute to the same end, the achievement of ideal conditions

The high flow rates found under these circumstances can therefore not be expected to appear under normal conditions, even if the air is saturated with humidity and of a constant high temperature as in the bronchi because a certain action of inhaled agents, gaseous or solid, on the epithelium and the mucous layer can never be excluded (Tremer *et al*, 1959)

*Conclusions* A group of tracheostomized patients has been studied with the author's technique and the mucus flow rate has been found to be relatively constant and high, approximately 10 mm per minute, regardless of age, sex and ambient relative hu-

*Table 8 Tracheostomy group Flou rates and corresponding relative humidity values before and/or after*

| Flow rate/Relative humidity | | | | | | |
|---|---|---|---|---|---|---|
| Case | 72 | 74 | 105 | 117 | 129 | 130 |
| Pre-op | | 0/31 | 0/34 | 6/30 | | 1 1/27 |
| Post op | | | | | | |
| 1 st week | 0/36 | 8/30 | 2/34 | 2 4/30 | 0/25 | |
| 2nd week | | 10/26 | 0/33 | 8/27 | 7 1/27 | 0/27 |
| | | | 4/32 | | 8/28 | 3/36 |
| | | | 8/30 | | | |
| 3rd week | 4 8/29 | | | | | 2 4/16 |
| | | | | | | 3/20 |
| | | | | | | 8/27 |
| 4th week | 8 8/27 | | 10/27 | | | |
| 5th-6th weeks | 12/35 | | 10/20 | | | 8/27 |

midity The present results are in good agreement with earlier investigations the difference between flow rate means in normal and tracheostomized subjects is, how ever, even more marked in the present study

*Postoperative time factor* No previous attempts have been made to evaluate the time factor in the postoperative period, during which the flow rate increases to the high 10 mm per minute level The present technique offers a possibility of doing so at repeated intervals without disturbing the normal physiology of the mucosa

*Present material* The same area was observed at different times before and/or after tracheostomy or laryngectomy in a group of 11 cases (Table 8) Case No 219 is also included in 'flow rate study" group, Table 7, previously described All the other 10 cases have not been mentioned earlier In Table 8 the flow rate is marked with the relative humidity of the ambient air at the time of observation The mean values in the last column are plotted in a diagram (Fig 8) which has no pretensions to exactness but schematically demonstrates the steep increase in flow rate during the second and third postoperative weeks After four weeks almost no further increase takes place

*Comments* During the first postoperative week there is an abundance of nasal secretions which cover the boggy bluish grey mucosa even in those cases that showed dry crusts before surgery During this period the flow is either sluggish or completely absent, probably owing to an excess amount of mucus on which not even gravity seems to have any influence

During the second and third weeks, previously inactive areas on the septum and the anterior ends of the turbinates begin to show signs of ciliary activity, *i e* flickering

*operation The mean values of all examinations during the corresponding week are given in the last column*

| 139 | 165 | 167 | 186 | 219 | Mean |
|---|---|---|---|---|---|
| 4 4/27 | | | 0/30 | 5 5/60 | 2 4/34 |
| | | | | | |
| 0/17 | 5 5/35 | | 6/41 | | 2 7/33 |
| 4/20 | 6/38 | 8/39 | 6/52 | 6 7/60 | |
| | | | | 10 9/48 | 6 0/34 |
| | | | | | |
| | | 10/25 | | | |
| | | | | | 6 7/24 5 |
| | | | | | |
| | | | | 12/72 | 10 0/43 |
| | | | | 12/40 | 10 5/30 5 |

light reflexes, caused by groups of cilia beating in approximately the same phase During this period the amount of mucus gradually diminishes and the flow rate on the septum increases

Within three or four weeks the active areas have spread to the very tips of the middle and inferior turbinates and to the most anterior part of the septum, close to the vestibule The flow in these areas usually goes in a dorsocaudal direction on the septum and straight backwards or slightly downwards on the turbinates Once established, the flow rate and direction remain stationary as long as the patient is tracheo stomized

*Conclusions* In a group of 11 subjects, observed before and/or after tracheostomy or laryngectomy, the mucus flow rate increased from a mean of 2 4 mm per minute before operation to 10 5 mm per minute after four to five weeks, the steepest increase taking place during the second and third postoperative weeks, during which period ciliary activity was seen to appear on previously inactive areas The obtained values are in good agreement with the results from other parts of the present investigation

*Decannulation* The assumption that the increase in flow rate is brought about by shunting the respiratory air stream away from the nose necessitates a special study of the flow rate after re routing the air through the nose by closing the tracheostomy wound This has not been done previously, owing to the lack of a suitable technique of observation

*Present material* Three patients, cases No 74, 161 and 167, tracheostomized for more than three weeks were observed before and after decannulation (Fig 9)

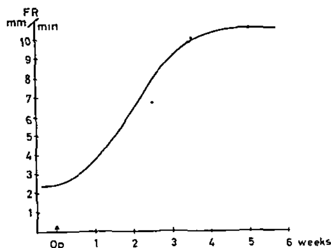



Fig 8 Schematic diagram of the increase in flow rate after tracheostomy (Op)

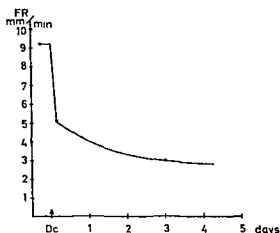



Fig 9 Schematic diagram of the decrease in flow rate after decannulation (Dc)

The mean flow rate, while breathing through the cannula, was 9 2 mm per minute and the mean time after tracheostomy was 24 days The mean rate two to three days after decannulation was 3 0 mm per minute at a relative humidity of 40 per cent, but already after three hours the mean rate had decreased to 5 1 mm per minute at 33 per cent relative humidity

*Comments* The rapid decrease noticed even during the first hours after decannulation, before histological changes could be the prime factor, favours the assumption that evaporation, changes in viscosity and drying of the secretions play the most important part in this connection

*Conclusions* In three subjects, tracheostomized for more than three weeks, the mucus flow rate mean decreased from 9 2 mm per minute to 3 0 mm per minute within a few days after decannulation Although the group is too small for statistical




Fig 10 Specimen No 1 5 days postoperatively Electron micrograph 6 000 ×
*Cy* Cylindrical cell, without cilia *F* Microvilli or filiform projections *G* Goblet cell *PnC* Polynuclear white blood cell

analysis, the tendency is uniform and evident a rapid decrease, probably caused by the drying effect of the respiratory air stream previously demonstrated in the present investigation

## Morphological changes

*Earlier studies* Somewhat controversial results have been obtained by different authors regarding the histology of the normal nasal mucosa and the changes observed after tracheostomy

Most authors agree on finding a non ciliated squamous epithelium on the anterior ends of the inferior and middle turbinates in normal subjects and attribute this metaplasia to a constant irritation by non conditioned inspiratory air (Oppikofer, 1907 Hilding, 1931a, 1932a, 1960 Dixon *et al*, 1949)

Leading the respiratory air away from the nose results in mucosal changes in almost all cases, the one most often observed is a marked increase in the number of ciliated and goblet cells (Sternberg 1924 Hilding, 1931c, 1932c) Dixon *et al* (1949), by




Fig 11 a Specimen No 2 7 days postoperatively Electron micrograph 6 000 ×
*C* Some scattered cilia, about 3 microns long *M* Mucous layer, resting on the tips of the cilia The arrow points at one of the centrioles or basal bodies, from which cilia are thought to develop

comparing specimens obtained from the anterior end of the inferior turbinate of normal and laryngectomized subjects, found the squamous epithelium to change into a columnar and well ciliated one but did not consider the goblet cells to predominate over the ciliated ones Schwab (1955) in a similar study, however, stated that even under normal circumstances were the anterior ends of the turbinates covered with a ciliated epithelium and that laryngectomy did not change the epithelium in this respect All these authors agree, however, on finding a ciliated columnar epithelium, rich in goblet cells and with minimal signs of inflammation on the anterior ends of the turbinates after laryngectomy In an extensive study on the regeneration of the nasal mucosa in lambs, Boling (1935) found cilia to regenerate in approximately two weeks after local trauma Burian (1960) found a complete regeneration with cilia to occur in about eight to ten weeks after extensive and deep damage to the nasal mucosa in rats

*Present study* As has been stated, signs of ciliary activity were seen to appear on previously inactive areas on the anterior ends of the inferior and middle turbinates at about the same time as the steepest increase in flow rate on the septum occurred




Fig. 11 b. Specimen No. 2, 7 days postoperatively. Electron micrograph 18 000.
*B* Basal body, the one at extreme left showing typical cross striation at the place of the rootlet. *C* Cilia about 3 microns in length and 0.2 microns in diameter. *F* Filiform projections. *M* Mucous layer 2.5 to 3 microns above the epithelial surface.

i.e. during the second and third postoperative weeks. In order to investigate whether a known inactive area may develop a mature ciliated epithelium in such a short period of time, repeated biopsies were taken from an area not showing any signs of ciliary activity before laryngectomy.

*Material.* Case 106 was a 48 years old man with a laryngeal carcinoma, non smoker but with a history of exposition to $SO_2$ fumes for many years. He was first examined before laryngectomy and showed a dry nasal mucosa with crust formation and no signs of ciliary activity on the anterior two cm of the middle turbinate and the usual septal area. The relative humidity levels at the time of the study varied between 20 per cent and 35 per cent. The patient was examined on different occasions following laryngectomy (Table 8), during which time the previously dry mucosa became moist and the mucus flow rate on the septum increased from 0 to 10 mm per minute in three weeks. The case was considered to be representative for the planned study and the patient gave his permission after having been informed of the purpose of the investigation.



Fig 12 Specimen No 3 11 days postoperatively Electron micrograph 6,000 ×
*C* Cilia, increasing in length and number as compared with specimen No 2 *G* Goblet cell

Four biopsies were taken from the anterior part of the left middle turbinate in an area that showed no signs of ciliary activity before the laryngectomy No anaesthesia was used

*Methods* The specimens were fixed in 1 per cent buffered osmium tetroxide solution, dehydrated in alcohol and embedded in Epon 1 micron thick sections were stained with toluidine blue, buffered to pH 9 and observed in an Ortholux Leitz microscope Thin sections were cut with a LKB Ultratome, stained with uranyl acetate and lead hydroxide and studied in a Siemens Elmiskop I electron muscroscope

*Results* Specimen No 1 (Fig 10) from the anterior tip, taken on the 5th postoperative day, was covered with an irregular stratified epithelium The surface layer consisted of cubical and cylindrical cells alternating with goblet cells The cylindrical and cubical cells were provided with microvilli but only scattered cells with cilia were seen, mostly in epithelial crypts

Specimen No 2 (Fig 11a, b) taken 10 mm from the anterior tip on the inferior margin on the 7th postoperative day demonstrated an entirely different picture The epithelium was of a regular cylindrical stratified type and only a few goblet cells were




Fig 13 Specimen No 4 16 days postoperatively Electron micrograph 6 000 ×
*C* Cilia which are mature and appear as a dense carpet on the surface *V* Vacuoles in the protoplasm of the ciliated cell

seen The surface was covered with a mucous layer located about three microns above the surface of the cells In light microscopy scattered tufts of short cilia were observed Electron microscopy demonstrated that the majority of surface cells contained a large number of formations in the area below the surface plasma membrane identical in structure with the basal bodies of normal kinocilia Some cells had no cilia at all, while others were provided with short cilia of varying length but never longer than four microns Even very short cilia barely protruding from the cellular surface were sometimes seen

Specimen No 3 (Fig 12), taken some few mm in front of specimen No 2 11 days postoperatively was covered with a regular cylindrical epithelium of the respiratory type Several goblet cells were seen between the cylindrical surface cells which were covered with tufts of cilia about four microns in length Some shorter cilia but only scattered basal bodies without connection with cilia were observed

In specimen No 4 (Fig 13), taken some mm behind specimen No 1, 16 days after laryngectomy typical respiratory tract epithelium was found Some goblet cells were seen and the cylindrical surface cells were covered with long dense tufts of cilia, approximately seven microns in length Large secretion vacuoles were observed in the bodies of the ciliated cells

*Comments* The previously mentioned studies by Sternberg, Dixon *et al* and Schwab were made several years after laryngectomy and there are no reports in the literature on the histological changes in the nasal mucosa during the immediate postoperative period in humans

Although the present investigation is based only on specimens from a single subject, it is clearly demonstrated that the epithelial changes may occur very soon after laryngectomy A regular cylindrical epithelium appears on the anterior end of the middle turbinate after only seven days At the same time the cylindrical cells contain a large number of basal bodies, presumably being the first sign of developing cilia Only a few cells have scattered cilia extending from their surface and these cilia have not reached full length

After eleven days most of the cells are covered with cilia, which are, however, shorter than in the mature epithelium Sixteen days after laryngectomy a mature epithelium with a dense ciliary population of normal length has developed

The goblet cells are very scarce in the first two specimens but increase in number during the second postoperative week At the same time intracellular vacuoles start to appear in the ciliated cells, but they are not fully developed until after sixteen days Sternberg stated that the goblet cells dominated over the ciliated cells in the nose of laryngectomized patients, a finding that was not supported by Dixon *et al* This discrepancy in opinions is probably explained by the fact that the ciliated cells became so filled with vacuoles that they may have been mistaken for goblet cells

*Conclusions* The morphological study has demonstrated that a previously inactive non ciliated epithelium may change into an active and ciliated cylindrical type, rich in goblet cells during a period of about two weeks under ideal conditions It can be reasonably assumed that similar changes occur in the septal mucosa contributing to the increase in flow rate occurring during the same time

# General Discussion

Although the results are obtained from a limited area in the nose and different flow rates may be found in other parts of the respiratory tract, there is no reason to believe that the ciliated mucosa in other areas would behave in a fundamentally different way Dry air is known to inflict damage to the cilia, but there are no earlier human *in vivo* studies The findings in the present investigation corroborate the earlier reports, stating the 70 per cent relative humidity level as an approximate borderline between harmful and harmless action on the flow rate

*In the present study relative humidity seems to be the most influential factor on the flow-rate variability* The underlying mechanism cannot be elucidated by the used method, only dealing with the flow rate as a total but, hypothetically, low relative humidity can be assumed to cause quantitative as well as qualitative changes in the mucous layer beyond the compensating ability of the mucosa, resulting in for instance, highly viscous secretions and/or a reduced amount of secretions, only moved with great difficulties by the cilia, ultimately reducing the beat rate More accentuated drying eventually leads to ciliary damage and death, the epithelium changing to a squamous type This metaplasia is, however, reversible as has been shown in the tracheostomy group, where a ciliated epithelium regenerated within three weeks after ideal conditions had been established

The high dispersion in flow rate in the normal material, particularly at lower humidities is probably caused by such changes in the mucosa and the mucous layer owing to anatomical differences, bringing the non conditioned air in a more or less direct contact with the observation area The present technique does not offer possibilities to vary the location of this area to a greater extent as the dorso-caudal flow is restricted to a relatively small surface If feasible determination of the mean flow rate from a number of different observation areas would possibly have reduced this dispersion, but the present results give a more truthful picture of the actual behaviour of the mucus flow under the influence of external factors

*If the mean flow rate value at 100 per cent relative humidity is extrapolated from* the normal group in Fig 7, 7—9 mm per minute will be obtained, a figure in relatively good agreement with the mean flow rate in the tracheostomy group The highest individual flow rate values in the present investigation, 12 mm per minute, were found in both the normal and the tracheostomy groups This favours the assumption that conditions in a limited area of the normal nasal mucosa, sheltered from harmful impingement by non-conditioned air, are similar to those in the nose of a tracheostomized patient as regards the ciliary and mucous factors *The optimal flow rate mean in the anterior part of the septal mucosa would thus be approximately 10 mm per minute, with a maximum of about 13 mm per minute*

An overall high flow rate over a larger area in the nose cannot, however, be obtained in a normal subject by simply elevating the relative humidity of the ambient air immediately prior to and during the observation time, as there are certain areas which have a reduced ciliary activity due to long-standing exposure to non conditioned air *The optimal conditions must be prevalent for a considerable time so that these areas may develop an optimal coating of ciliated cells before the high and constant flow rate is reached Under ideal conditions such a process has been observed to occur in about two weeks If the present technique should be used for studying the influence of certain factors on the flow rate, such as tobacco smoke, air pollutants, drugs etc, the basic flow rate for the specific area must therefore first be determined for reference*

The significant differences in flow rates between smokers and non smokers, especially at lower relative humidity levels, cannot be explained by the present investigation Hypothetically, however, the combined effect exerted on the cilia and the autonomically innervated mucosal elements by the various components of tobacco smoke may conceivably cause changes in the different factors regulating the mucus flow A quantitative or qualitative change in the mucous layer, already affected by a low relative humidity, will certainly produce a more marked decrease in flow rate than would a similar change under more optimal conditions The results are in no respects directly applicable to the lower respiratory tract, where smoke in considerably higher concentrations is inhaled and filtered and the components, entrapped or dissolved in the mucous layer, may directly affect the mucosa for a longer time It is, however, remarkable that mucus flow may be so obviously influenced by smoking, even when direct action on the mucosa, at least to a great extent, can be excluded, a fact that indicates that the reduced flow rate mean in the smokers is, at least partly, a general effect of tobacco smoke

# Summary

A quantitative method was developed by the author for observation of the mucus flow rate in the human nose. The technique was based on direct observation under magnification of the mucus flow, indicated by a tracer substance with exact measurement of the time and distance travelled by the substance and maintenance of physiological conditions.

The evolved technique was used on a normal series in 174 examinations, done at varying relative humidity levels but under otherwise constant conditions with the following results. The mean flow rate was 4.2 mm per minute at 43.6 per cent relative humidity. The flow rate was significantly correlated to the relative humidity of the ambient air ($P<0.001$). Above 70 per cent relative humidity mucus flow could be seen in all the observed cases, but below that level cessation of the flow was observed in an increasing number of cases. No statistically significant differences could be related to variations in sex and age. Smokers had a statistically significant lower flow rate mean than the non-smokers at 43.6 per cent relative humidity ($P<0.001$), 3.6 mm per minute and 4.8 mm per minute respectively. At higher humidity levels the difference between the groups seemed to diminish.

In a special study on tracheostomized patients the flow rate under constant and optimal conditions was investigated. The mean flow rate was 10.4 mm per minute regardless of age and sex and the relative humidity. The results indicate the deleterious effect of non-conditioned air on ciliary clearance and corroborate the findings obtained in the study of the normal group that age and sex *per se* are without effect on the flow rate. In order to evaluate changes occurring in a normal nasal mucosa exposed to optimal conditions, a group of patients were observed before and/or after tracheostomy. The steepest increase in flow rate took place during the second and third postoperative weeks and the functional changes proceeded parallel with the morphological ones as observed with light and electron microscopy.

The present investigation has thus shown the fundamental influence of the relative humidity of the respiratory air on the mucus flow rate in the nose, an overall optimal flow rate only obtainable in saturated air. A general high flow rate presupposes *inter alia* a generally well-developed ciliary epithelium and cannot be expected to occur by simply elevating the ambient relative humidity during the time of examination. Consequently the basic flow rate for a specific area must be determined and used for comparison, when the method is used for studies of the action of certain agents on the nasal mucus flow rate.

The method developed by the author was found to be well suitable for repeated observations of the nasal mucus flow rate in humans under variable but physiological conditions and did not cause any discomfort to the subject, thus meeting the requirements for a human *in vivo* technique.

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Tables 9—17

*Table 9 Normal group Relative humidity less than 40 per cent Age 31—60 years*

| Case | RH % | Age | Sex | Smoking habits | Fast side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Slow side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 151 | 20 | 33 | ♂ | M | ∞ | | | ∞ | 0.0 | ∞ | | | ∞ | 0.0 | 0.0 |
| 152 | 20 | 49 | ♀ | N | 25 | 25 | | 25 | 4.8 | 25 | 25 | | 25 | 4.8 | 4.8 |
| 153 | 20 | 54 | ♀ | N | ∞ | | | ∞ | 0.0 | ∞ | | | ∞ | 0.0 | 0.0 |
| 97 | 23 | 33 | ♂ | N | 30 | 30 | 30 | 30 | 4.0 | 36 | 36 | 36 | 36 | 3.3 | 3.6 |
| 141 | 23 | 33 | ♂ | M | 17 | 17 | 17 | 17 | 7.1 | ∞ | | | ∞ | 0.0 | 3.6 |
| 70 | 23 | 42 | ♀ | M | 35 | 35 | 35 | 35 | 3.4 | ∞ | | | ∞ | 0.0 | 1.7 |
| 68 | 23 | 43 | ♀ | M | 40 | 40 | 40 | 40 | 3.0 | ∞ | | | ∞ | 0.0 | 1.5 |
| 87 | 25 | 36 | [illegible] | N | 25 | 25 | 25 | 25 | 4.8 | 25 | 25 | 25 | 25 | 4.8 | 4.8 |
| 158 | 26 | 40 | ♂ | M | ∞ | | | ∞ | 0.0 | ∞ | | | ∞ | 0.0 | 0.0 |
| 148 | 27 | 50 | ♀ | N | ∞ | | | ∞ | 0.0 | ∞ | | | ∞ | 0.0 | 0.0 |
| 130 | 27 | 56 | ♂ | M | 100 | 100 | 100 | 100 | 1.1 | 100 | 110 | 120 | 110 | 1.1 | 1.1 |
| 154 | 28 | 33 | ♂ | H | ∞ | | | ∞ | 0.0 | ∞ | | | ∞ | 0.0 | 0.0 |
| 156 | 28 | 33 | ♀ | N | 35 | 35 | | 35 | 3.4 | ∞ | | | ∞ | 0.0 | 1.7 |
| 149 | 28 | 34 | ♀ | H | 50 | 50 | 50 | 50 | 2.4 | ∞ | | | ∞ | 0.0 | 1.2 |
| 155 | 28 | 38 | ♂ | N | 12 | 13 | 12 | 12 | 10.0 | 12 | 12 | 12 | 12 | 10.0 | 10.0 |
| 157 | 28 | 45 | ♂ | N | 15 | 15 | 15 | 15 | 8.0 | 15 | 15 | 15 | 15 | 8.0 | 8.0 |
| 136 | 28 | 48 | ♀ | N | 17 | 17 | 17 | 17 | 7.1 | ∞ | | | ∞ | 0.0 | 3.6 |
| 143 | 28 | 50 | ♀ | N | 12 | 12 | 12 | 12 | 10.0 | 12 | 12 | 12 | 12 | 10.0 | 10.0 |
| 147 | 28 | 53 | ♀ | N | ∞ | | | ∞ | 0.0 | ∞ | | | ∞ | 0.0 | 0.0 |
| 146 | 28 | 55 | ♀ | M | 60 | 60 | | 60 | 2.0 | ∞ | | | ∞ | 0.0 | 1.0 |
| 144 | 28 | 57 | ♀ | N | 10 | 10 | 10 | 10 | 12.0 | 20 | 20 | 20 | 20 | 6.0 | 9.0 |
| 100 | 29 | 32 | ♀ | M | 32 | 32 | 32 | 32 | 3.8 | ∞ | | | ∞ | 0.0 | 1.9 |
| 101 | 29 | 33 | [illegible] | N | 32 | 32 | 32 | 32 | 3.8 | ∞ | | | ∞ | 0.0 | 1.9 |
| 99 | 29 | 52 | ♂ | M | 60 | 60 | | 60 | 2.0 | ∞ | | | ∞ | 0.0 | 1.0 |
| 93 | 29 | 53 | ♂ | M | 30 | 30 | 30 | 30 | 4.0 | 30 | 30 | 30 | 30 | 4.0 | 4.0 |
| 102 | 29 | 56 | ♀ | N | 20 | 22 | 22 | 21 | 5.7 | 22 | 23 | 20 | 22 | 5.5 | 5.6 |
| 132 | 30 | 36 | ♂ | H | ∞ | | | ∞ | 0.0 | ∞ | | | ∞ | 0.0 | 0.0 |
| 124 | 30 | 40 | ♂ | M | 60 | 100 | | 80 | 1.5 | ∞ | | | ∞ | 0.0 | 0.8 |
| 127 | 30 | 45 | | N | 15 | 15 | 15 | 15 | 8.0 | ∞ | | | ∞ | 0.0 | 4.0 |
| 125 | 30 | 55 | [illegible] | M | 17 | 17 | 17 | 17 | 7.1 | 40 | 40 | 40 | 40 | 3.0 | 5.0 |
| 123 | 30 | 58 | ♂ | M | 15 | 15 | 15 | 15 | 8.0 | ∞ | | | ∞ | 0.0 | 4.0 |
| 186 | 30 | 60 | ♂ | H | 20 | 20 | 20 | 20 | 6.0 | ∞ | | | ∞ | 0.0 | 3.0 |
| 121 | 31 | 38 | ♂ | N | 13 | 13 | 13 | 13 | 9.2 | 13 | 13 | 13 | 13 | 9.2 | 9.2 |
| 74 | 31 | 48 | | N | 30 | 32 | 30 | 31 | 3.9 | ∞ | | | ∞ | 0.0 | 2.0 |
| 75 | 32 | 44 | [illegible] | N | 20 | 20 | 20 | 20 | 6.0 | 40 | 45 | 40 | 42 | 2.9 | 4.4 |
| 119 | 33 | 40 | [illegible] | H | 30 | 35 | | 33 | 3.6 | ∞ | | | ∞ | 0.0 | 1.8 |
| 118 | 33 | 54 | ♀ | M | 30 | 30 | 30 | 30 | 4.0 | ∞ | | | ∞ | 0.0 | 2.0 |
| 110 | 34 | 34 | ♂ | H | 18 | 18 | 18 | 18 | 6.7 | 50 | 50 | 50 | 50 | 2.4 | 4.6 |
| 103 | 34 | 43 | | H | ∞ | | | ∞ | 0.0 | ∞ | | | ∞ | 0.0 | 0.0 |
| 114 | 34 | 45 | ♂ | H | 33 | 33 | 33 | 33 | 3.6 | ∞ | | | ∞ | 0.0 | 1.8 |
| 106 | 34 | 48 | ♂ | N | 33 | 32 | 32 | 32 | 3.8 | ∞ | | | ∞ | 0.0 | 1.9 |

*Table 9 Cont*

| Case | RH % | Age | Sex | Smoking habits | Fast side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Slow side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 108 | 34 | 52 | ♂ | N | 30 | 30 | 30 | 30 | 4 0 | ∞ | | | ∞ | 0 0 | 2 0 |
| 112 | 34 | 52 | ♂ | H | 15 | 15 | 15 | 15 | 8 0 | 15 | 15 | 15 | 15 | 8 0 | 8 0 |
| 107 | 34 | 60 | ♂ | N | 30 | 30 | 30 | 30 | 4 0 | ∞ | | | ∞ | 0 0 | 2 0 |
| 71 | 36 | 32 | ♂ | N | 16 | 16 | 20 | 17 | 7 1 | ∞ | | | ∞ | 0 0 | 3 6 |
| 216 | 38 | 44 | ♂ | N | 20 | 20 | 20 | 20 | 6 0 | 20 | 20 | 20 | 20 | 6 0 | 6 0 |

*Table 10 Normal group Relative humidity less than 40 per cent Age 61 years and older*

| Case | RH % | Age | Sex | Smoking habits | Fast side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Slow side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 88 | 26 | 80 | ♀ | N | 40 | 45 | 45 | 43 | 2 8 | ∞ | | | ∞ | 0 0 | 1 4 |
| 81 | 27 | 69 | ♂ | M | 35 | 35 | 35 | 35 | 3 4 | 35 | 35 | 35 | 35 | 3 4 | 3 4 |
| 145 | 28 | 72 | ♀ | N | ∞ | | | ∞ | 0 0 | ∞ | | | ∞ | 0 0 | 0 0 |
| 134 | 30 | 63 | ♂ | N | 27 | 27 | 27 | 27 | 4 4 | 37 | 37 | 37 | 37 | 3 2 | 3 8 |
| 136 | 30 | 64 | ♀ | M | ∞ | | | ∞ | 0 0 | ∞ | | | ∞ | 0 0 | 0 0 |
| 126 | 30 | 65 | ♀ | N | 50 | 50 | 50 | 50 | 2 4 | ∞ | | | ∞ | 0 0 | 1 2 |
| 135 | 30 | 67 | ♀ | N | 15 | 15 | 15 | 15 | 8 0 | 27 | 27 | 26 | 27 | 4 4 | 6 2 |
| 117 | 30 | 67 | ♂ | H | 20 | 20 | 20 | 20 | 6 0 | ∞ | | | ∞ | 0 0 | 3 0 |
| 93 | 33 | 65 | ♂ | N | 25 | 25 | 25 | 25 | 4 8 | ∞ | | | ∞ | 0 0 | 2 4 |
| 94 | 34 | 62 | ♂ | N | 25 | 25 | 25 | 25 | 4 8 | 35 | 35 | 35 | 35 | 3 4 | 4 1 |
| 96 | 34 | 72 | ♂ | H | 30 | 30 | 30 | 30 | 4 0 | ∞ | | | ∞ | 0 0 | 2 0 |
| 85 | 35 | 61 | ♂ | H | 32 | 33 | 33 | 33 | 3 6 | 100 | 100 | | 100 | 1 2 | 2 4 |
| 217 | 38 | 68 | ♂ | N | 50 | 50 | 50 | 50 | 2 4 | ∞ | | | ∞ | 0 0 | 1 2 |

*Table 11 Normal group Relative humidity 40—59 per cent Age 30 years and younger*

| Case | RH % | Age | Sex | Smoking habits | Fast side: Time s/2 mm, Observation I | II | III | Mean | FR mm/min | Slow side: Time s/2 mm, Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 63 | 40 | 19 | ♂ | H | 15 | 15 | 15 | 15 | 8.0 | ∞ | | | ∞ | 0.0 | 4.0 |
| 64 | 40 | 25 | ♀ | N | 40 | 40 | 40 | 40 | 3.0 | ∞ | | | ∞ | 0.0 | 1.5 |
| 67 | 40 | 25 | ♀ | H | 12 | 12 | 12 | 12 | 10.0 | 22 | 20 | 20 | 21 | 5.7 | 7.8 |
| 69 | 40 | 25 | ♀ | N | 17 | 15 | 17 | 16 | 7.5 | 17 | 17 | 17 | 17 | 7.1 | 7.3 |
| 168 | 41 | 18 | ♀ | H | 30 | 30 | 30 | 30 | 4.0 | 30 | 30 | 30 | 30 | 4.0 | 4.0 |
| 191 | 45 | 14 | ♂ | N | 25 | 25 | 25 | 25 | 4.8 | ∞ | | | ∞ | 0.0 | 2.4 |
| 61 | 45 | 21 | ♀ | N | 20 | 20 | 20 | 20 | 6.0 | 20 | 20 | 20 | 20 | 6.0 | 6.0 |
| 159 | 45 | 22 | ♀ | N | 35 | 35 | 35 | 35 | 3.4 | 35 | 35 | 35 | 35 | 3.4 | 3.4 |
| 173 | 45 | 22 | ♀ | H | 25 | 25 | 25 | 25 | 4.8 | ∞ | | | ∞ | 0.0 | 2.4 |
| 169 | 45 | 23 | ♀ | N | 25 | 25 | 25 | 25 | 4.8 | 50 | 55 | 50 | 52 | 2.3 | 3.6 |
| 65 | 45 | 28 | ♀ | M | 22 | 20 | 21 | 21 | 5.7 | 20 | 22 | 21 | 21 | 5.7 | 5.7 |
| 176 | 45 | 25 | ♂ | N | 17 | 17 | 17 | 17 | 7.1 | 20 | 20 | 20 | 20 | 6.0 | 6.6 |
| 204 | 50 | 16 | ♀ | N | 22 | 22 | 22 | 22 | 5.5 | 25 | 25 | | 25 | 4.8 | 4.6 |
| 183 | 50 | 19 | ♀ | M | 25 | 25 | 25 | 25 | 4.8 | ∞ | | | ∞ | 0.0 | 2.4 |
| 184 | 50 | 20 | ♂ | N | 15 | 17 | 15 | 16 | 7.5 | 35 | 30 | | 32 | 3.8 | 5.6 |
| 8 | 50 | 26 | ♀ | N | 18 | 18 | 18 | 18 | 6.7 | ∞ | | | ∞ | 0.0 | 3.4 |
| 24 | 50 | 30 | ♀ | H | 22 | 22 | | 22 | 5.5 | 22 | 22 | | 22 | 5.5 | 5.5 |
| 203 | 52 | 21 | ♀ | N | 19 | 19 | 19 | 19 | 6.3 | ∞ | | | ∞ | 0.0 | 3.2 |
| 58 | 52 | 25 | ♂ | H | 15 | 17 | 17 | 16 | 7.5 | ∞ | | | ∞ | 0.0 | 3.8 |
| 59 | 52 | 25 | ♂ | N | 25 | 25 | 27 | 26 | 4.6 | 28 | 30 | 30 | 29 | 4.1 | 4.4 |
| 13 | 55 | 26 | ♀ | N | 15 | 15 | 16 | 15 | 8.0 | 25 | 23 | 25 | 24 | 5.0 | 6.5 |

*Table 12. Normal group. Relative humidity 40—59 per cent Age 31—60 years*

| Case | RH % | Age | Sex | Smoking habits | Fast side: Time s/2 mm Observation I | II | III | Mean | FR mm/min | Slow side: Time s/2 mm Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 70 | 40 | 42 | ♀ | M | 20 | 22 | 20 | 21 | 5 7 | ∞ | | | ∞ | 0 0 | 2 8 |
| 68 | 40 | 43 | ♀ | M | 15 | 15 | 15 | 15 | 8 0 | 15 | 15 | 15 | 15 | 8 0 | 8 0 |
| 62 | 40 | 56 | ♂ | N | 25 | 25 | 25 | 25 | 4 8 | 25 | 25 | 25 | 25 | 4 8 | 4 8 |
| 149 | 41 | 34 | ♀ | H | 30 | 30 | | 30 | 4 0 | 40 | 40 | 40 | 40 | 3 0 | 3 5 |
| 163 | 41 | 46 | ♀ | N | 17 | 17 | 17 | 17 | 7 1 | ∞ | | | ∞ | 0 0 | 3 6 |
| 196 | 41 | 51 | ♂ | N | 40 | 40 | 40 | 40 | 3 0 | ∞ | | | ∞ | 0 0 | 1 5 |
| 179 | 41 | 56 | ♀ | N | 25 | 25 | 25 | 25 | 4 8 | ∞ | | | ∞ | 0 0 | 2 4 |
| 180 | 41 | 56 | ♀ | N | 27 | 27 | 27 | 27 | 4 4 | 27 | 27 | 27 | 27 | 4 4 | 4 4 |
| 195 | 41 | 57 | ♀ | N | 17 | 17 | 17 | 17 | 7 1 | 22 | 22 | 22 | 22 | 5 5 | 6 8 |
| 164 | 42 | 46 | ♀ | N | 20 | 20 | 20 | 20 | 6 0 | 20 | 20 | 20 | 20 | 6.0 | 6 0 |
| 174 | 45 | 34 | ♂ | N | 30 | 30 | 30 | 30 | 4 0 | ∞ | | | ∞ | 0 0 | 2 0 |
| 162 | 45 | 37 | ♀ | M | 20 | 20 | 20 | 20 | 6 0 | 35 | 35 | | 35 | 3 4 | 4 7 |
| 193 | 45 | 43 | ♂ | N | 25 | 25 | 25 | 25 | 4 8 | 30 | 30 | 30 | 30 | 4 0 | 4 4 |
| 194 | 45 | 52 | ♂ | N | 20 | 20 | 20 | 20 | 6 0 | 30 | 30 | 30 | 30 | 4 0 | 5 0 |
| 160 | 45 | 55 | ♀ | N | 40 | 40 | | 40 | 3 0 | ∞ | | | ∞ | 0 0 | 1 5 |
| 170 | 45 | 60 | ♂ | M | 34 | 34 | 34 | 34 | 3 5 | 36 | 35 | 36 | 36 | 3 3 | 3 4 |
| 189 | 49 | 52 | ♀ | N | 12 | 12 | 12 | 12 | 10 0 | 12 | 12 | 12 | 12 | 10 0 | 10 0 |
| 188 | 49 | 56 | ♀ | M | 40 | 40 | 40 | 40 | 3 0 | 40 | 40 | 40 | 40 | 3 0 | 3 0 |
| 182 | 50 | 32 | ♀ | N | 30 | 33 | 30 | 31 | 3 9 | 50 | 55 | 55 | 53 | 2 3 | 3 1 |
| 141 | 50 | 33 | ♂ | M | 16 | 16 | 16 | 16 | 7 5 | 30 | 30 | 30 | 30 | 4 0 | 5 8 |
| 154 | 50 | 33 | ♂ | H | 22 | 22 | 22 | 22 | 5 5 | ∞ | | | ∞ | 0 0 | 2.8 |
| 158 | 50 | 41 | ♂ | M | 30 | 30 | 30 | 30 | 4 0 | ∞ | | | ∞ | 0 0 | 2 0 |
| 207 | 50 | 41 | ♂ | M | 23 | 23 | 22 | 23 | 5 2 | ∞ | | | ∞ | 0 0 | 2 6 |
| 209 | 50 | 47 | ♀ | N | 20 | 20 | 20 | 20 | 6 0 | ∞ | | | ∞ | 0 0 | 3 0 |
| 208 | 50 | 58 | ♀ | N | 12 | 12 | 12 | 12 | 10 0 | 12 | 12 | 12 | 12 | 10 0 | 10 0 |
| 199 | 52 | 40 | ♂ | H | 35 | 35 | 35 | 35 | 3 4 | ∞ | | | ∞ | 0 0 | 1 7 |
| 60 | 55 | 50 | ♀ | H | 17 | 17 | 17 | 17 | 7 1 | 17 | 17 | 17 | 17 | 7 1 | 7 1 |
| 215 | 56 | 31 | ♀ | N | 20 | 20 | 20 | 20 | 6 0 | 30 | 30 | 30 | 30 | 4 0 | 5 0 |

*Table 13 Normal group Relative humidity 40—59 per cent Age 61 years and older*

| Case | RH % | Age | Sex | Smoking habits | Fast side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Slow side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 178 | 41 | 78 | ♀ | N | 35 | 35 | 35 | 35 | 3 4 | 35 | 35 | 35 | 35 | 3 4 | 3 4 |
| 172 | 45 | 63 | ♀ | N | 50 | 50 | 50 | 50 | 2 4 | ∞ | | | ∞ | 0 0 | 1 2 |
| 171 | 45 | 70 | ♂ | N | 20 | 20 | 20 | 20 | 6 0 | 50 | 50 | 60 | 55 | 2 2 | 4 1 |
| 192 | 45 | 74 | ♂ | M | 40 | 40 | 40 | 40 | 3 0 | ∞ | | | ∞ | 0 0 | 1 5 |
| 187 | 49 | 62 | ♀ | N | 12 | 12 | 12 | 12 | 10 0 | 12 | 12 | 12 | 12 | 10 0 | 10 0 |
| 181 | 50 | 67 | ♀ | N | 20 | 20 | 20 | 20 | 6 0 | 50 | 50 | | 50 | 2 4 | 4 2 |
| 206 | 50 | 78 | ♂ | M | 22 | 22 | 22 | 22 | 5 5 | ∞ | | | ∞ | 0 0 | 2 8 |
| 198 | 52 | 72 | ♂ | N | 25 | 25 | 25 | 25 | 4 8 | ∞ | | | ∞ | 0 0 | 2 4 |
| 201 | 53 | 67 | ♂ | H | 35 | 35 | 35 | 35 | 3 4 | 40 | 40 | 40 | 40 | 3 0 | 3 2 |
| 200 | 53 | 74 | ♂ | N | 20 | 20 | 20 | 20 | 6 0 | 25 | 25 | 25 | 25 | 4 8 | 5 4 |

*Table 14 Normal group Relative humidity 60—79 per cent Age 30 years and younger*

| Case | RH % | Age | Sex | Smoking habits | Fast side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Slow side Time s/2 mm Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 57 | 60 | 16 | ♂ | N | 12 | 11 | 11 | 11 | 10 9 | ∞ | | | ∞ | 0 0 | 5 4 |
| 233 | 62 | 24 | ♀ | H | 24 | 24 | 24 | 24 | 5 0 | 30 | 30 | 30 | 30 | 4 0 | 4 5 |
| 8 | 62 | 25 | ♀ | N | 18 | 18 | 18 | 18 | 6 7 | ∞ | | | ∞ | 0 0 | 3 4 |
| 77 | 63 | 25 | ♀ | N | 20 | 20 | 20 | 20 | 6 0 | 20 | 20 | | 20 | 6 0 | 6 0 |
| 232 | 63 | 28 | ♀ | N | 17 | 17 | 17 | 17 | 7 1 | 30 | 30 | 30 | 30 | 4 0 | 5 6 |
| 20 | 65 | 12 | ♂ | N | 10 | 10 | 10 | 10 | 12 0 | 11 | 11 | 10 | 11 | 10 9 | 11 4 |
| 228 | 70 | 12 | ♂ | N | 15 | 15 | 15 | 15 | 8 0 | 24 | 26 | 22 | 24 | 5 0 | 6 5 |
| 19 | 70 | 27 | ♀ | N | 13 | 12 | 11 | 12 | 10 0 | 25 | 22 | 25 | 24 | 5 0 | 7 5 |
| 24 | 70 | 29 | ♀ | M | 15 | 15 | 15 | 15 | 8 0 | 22 | 22 | | 22 | 5 5 | 6 8 |
| 226 | 70 | 29 | ♂ | N | 34 | 34 | 34 | 34 | 3 5 | 34 | 34 | 34 | 34 | 3 5 | 3 5 |

*Table 15 Normal group Relative humidity 60—79 per cent Age 31—60 years*

| Case | RH | Age | Sex | Smoking habits | Fast side: Time s/2 mm, Observation I | II | III | Mean | FR mm/min | Slow side: Time s/2 mm, Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 219 | 60 | 36 | ♂ | N | 13 | 13 | 13 | 13 | 9.2 | 22 | 22 | 22 | 22 | 5.5 | 7.4 |
| 221 | 60 | 41 | ♂ | M | 17 | 17 | 17 | 17 | 7.1 | 17 | 17 | 17 | 17 | 7.1 | 7.1 |
| 220 | 61 | 54 | ♀ | N | 17 | 17 | 17 | 17 | 7.1 | 17 | 17 | 17 | 17 | 7.1 | 7.1 |
| 154 | 62 | 33 | ♂ | H | 20 | 20 | | 20 | 6.0 | 40 | 40 | 40 | 40 | 3.0 | 4.5 |
| 214 | 62 | 45 | ♀ | N | 25 | 25 | 25 | 25 | 4.8 | 30 | 30 | 30 | 30 | 4.0 | 4.4 |
| 211 | 63 | 34 | ♀ | N | 18 | 18 | 18 | 18 | 6.7 | 25 | 25 | 25 | 25 | 4.8 | 5.8 |
| 213 | 63 | 59 | ♂ | H | 37 | 37 | 37 | 37 | 3.2 | 37 | 37 | 37 | 37 | 3.2 | 3.2 |
| 21 | 65 | 44 | ♂ | M | 16 | 16 | 15 | 16 | 7.5 | 17 | 17 | 16 | 17 | 7.1 | 7.3 |
| 22 | 65 | 48 | ♂ | N | 20 | 21 | 20 | 20 | 6.0 | 22 | 22 | 23 | 22 | 5.5 | 5.8 |
| 52 | 65 | 49 | ♂ | M | 13 | 14 | 15 | 14 | 8.6 | 20 | 20 | 20 | 20 | 6.0 | 7.3 |
| 235 | 70 | 33 | ♀ | H | 20 | 20 | 20 | 20 | 6.0 | 20 | 20 | 20 | 20 | 6.0 | 6.0 |
| 224 | 70 | 51 | ♂ | H | 13 | 13 | 13 | 13 | 9.2 | 15 | 15 | 15 | 15 | 8.0 | 8.6 |
| 234 | 70 | 53 | ♀ | H | 15 | 15 | 15 | 15 | 8.0 | 15 | 15 | 15 | 15 | 8.0 | 8.0 |

*Table 16 Normal group Relative humidity 60—79 per cent Age 61 years and older*

| Case | RH | Age | Sex | Smoking habits | Fast side: Time s/2 mm, Observation I | II | III | Mean | FR mm/min | Slow side: Time s/2 mm, Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 56 | 60 | 61 | ♂ | M | 17 | 17 | 20 | 18 | 6.7 | 22 | 22 | 20 | 21 | 5.7 | 6.2 |
| 212 | 63 | 64 | ♂ | M | 30 | 30 | 30 | 30 | 4.0 | 40 | 40 | | 40 | 3.0 | 3.5 |
| 222 | 63 | 71 | ♂ | M | 15 | 15 | 15 | 15 | 8.0 | 15 | 15 | 15 | 15 | 8.0 | 8.0 |
| 55 | 65 | 66 | ♂ | H | 22 | 20 | 19 | 20 | 6.0 | 22 | 22 | 25 | 23 | 5.2 | 5.6 |
| 231 | 67 | 62 | ♂ | N | 15 | 15 | 15 | 15 | 8.0 | 15 | 15 | 15 | 15 | 8.0 | 8.0 |
| 229 | 67 | 66 | ♂ | N | 10 | 10 | 10 | 10 | 12.0 | 10 | 10 | 10 | 10 | 12.0 | 12.0 |
| 50 | 67 | 69 | ♂ | N | 20 | 21 | 20 | 20 | 6.0 | 20 | 21 | 20 | 20 | 6.0 | 6.0 |
| 23 | 70 | 74 | | N | 22 | 21 | 21 | 21 | 5.7 | ∞ | | | ∞ | 0.0 | 2.8 |

*Table 17 Normal group Relative humidity 80 per cent and above Age 28—51 years*

| Case | RH % | Age | Sex | Smoking habits | Fast side: Time s/2 mm: Observation I | II | III | Mean | FR mm/min | Slow side: Time s/2 mm: Observation I | II | III | Mean | FR mm/min | Mean FR both sides |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 25 | 80 | 28 | ♀ | H | 14 | 14 | 13 | 14 | 8 6 | 25 | 25 | 25 | 25 | 4 8 | 6 7 |
| 26 | 80 | 28 | ♂ | N | 15 | 15 | 15 | 15 | 8 0 | 15 | 15 | 15 | 15 | 8 0 | 8 0 |
| 29 | 80 | 29 | ♂ | N | 15 | 15 | 15 | 15 | 8 0 | 20 | 20 | 20 | 20 | 6 0 | 7 0 |
| 27 | 80 | 34 | ♂ | N | 15 | 15 | 15 | 15 | 8 0 | 20 | 20 | 20 | 20 | 7 0 | 7 0 |
| 33 | 85 | 51 | ♂ | M | 15 | 17 | 17 | 16 | 7 5 | 20 | 20 | 20 | 20 | 6 0 | 6 8 |
| 37 | 88 | 33 | ♀ | N | 15 | 15 | 15 | 15 | 8 0 | 15 | 15 | 15 | 15 | 8 0 | 8 0 |

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# HE ENDOLYMPHATIC DUCT AND SAC IN THE GUINEA PIG

An electron microscopic
and experimental investigation

BY
PER-GOTTHARD LUNDQUIST

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

ACTA OTO-LARYNGOLOGICA
SUPPLEMENTUM 201

FROM KING GUSTAF V RESEARCH INSTITUTE AND THE DEPARTMENT OF OTOLARYNGOLOGY, KAROLINSKA SJUKHUSET, STOCKHOLM, SWEDEN

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STOCKHOLM 1965

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KUNGL. BOKTRYCKERIET P. A. NORSTEDT & SÖNER

*To My Wife*

## Contents

# Gross Anatomy of the Endolymphatic Duct and Sac

## *Introduction*

The inner ear consists of a series of thin walled sacs and ducts enclosed in the petrous temporal bone These are filled with a clear fluid, the endolymph The space between this thin-walled system and its bony walls is also filled with a clear fluid, the perilymph

Antonio Scarpa first described the human inner ear in 1789 comprising of two "stones sacs" and the membranous semicircular ducts and gave their position in the bone The two fluids endolymph and perilymph were described by Breschet in 1833

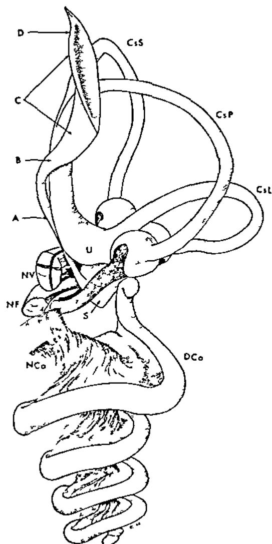
However, the membranous labyrinth is not completely enclosed in bone, one part, the endolymphatic sac protruding partially out of the petrous bone in close relation to the sigmoid sinus at the external aperture of the vestibular aqueduct

The vestibular aqueduct was discovered by Domenico Cotugno, who described its anatomy in his work "*De Aquaeductibus Auris Humanae Internae*" (1774) He noticed that a small intradural cavity '*cavitas aquaeductus membranea*", just beside the external aperture of the vestibular aqueduct was apparently connected to the vestibule By injecting mercury into the cochlear aqueduct he succeeded in also filling this intradural cavity, thus demonstrating its connection with the vestibule

*In 1869 Boettcher carried out a more detailed investigation on sheep embryos and* cats and was subsequently able to show the true relationship of this intradural cavity, to the membranous labyrinth He found that the membranous part of the vestibular aqueduct was continued into this sack-like appendage on the one hand, whilst on the other it was continuous with the utricle and saccule He also showed that the whole system was filled with endolymph, not perilymph as had been believed by earlier investigators It was not until 1873 that Hasse gave the 'endolymphatic duct and sac" to this appendage

The comparative anatomy of the endolymphatic duct and sac in the vertebrates, was first described by Retzius in 1881 but it was in 1900 that Alexander described its anatomy in guinea pig embryos and 1919 Portmann gave his account in the adult guinea pig (Fig 1)

However, it was Stacy Guild in 1927 who made the first complete study of the anatomy and histology of this system in the guinea pig In 1924 an excellent study of the complete labyrinthine system in the bat was published by Iwata Many investigators have described the anatomy and histology of the endolymphatic sac in [illegible]mans the most prominent being Siebenmann (1919), Sirala (1942), Secretan [illegible] F[illegible]trom (1951) Saxen (1951) and Bast and Anson (1949) The latter [illegible] pa[illegible] [illegible]culo[illegible]s account of the embryological development of the endolymphatic duc[illegible]



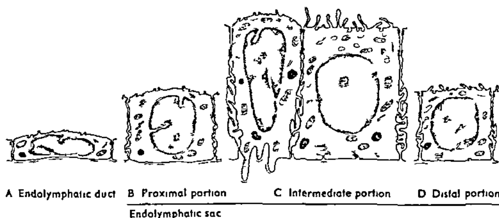




Fig 1 Schematic drawing of the endolymphatic duct and sac and the labyrinth from a posteromedial view The cells at the bottom of the picture illustrate the epithelial cell types found in the various parts of the endolymphatic duct and sac CsL CsP and CsS, lateral, posterior and superior semicircular canals DCo cochlear duct NCo cochlear nerve, NF, facial nerve, NV vestibular nerve S saccule U utricle Reconstructed from an adult guinea pig right side)

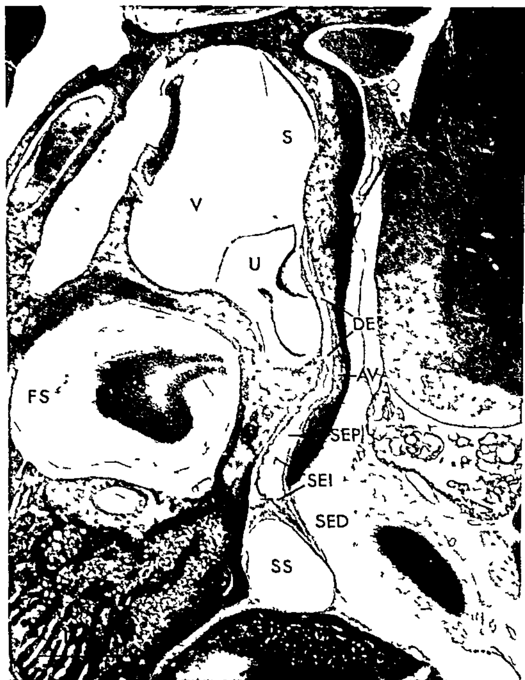



Fig 2 Low power (light microscopic) view of the endolymphatic duct (DE) which begins in the vestibule (V) at the confluence of the ducts from the saccule (S) and utricle (U) respectively It passes through the vestibular aqueduct (AV) and changes gradually into the widening first part of the endolymphatic sac the proximal portion (SEP) The following intermediate portion (SEI) is the most complex part and lies partly inside and partly outside the vestibular aqueduct The intermediate portion changes gradually to the flattened distal portion of the sac (SED) which is the termination of the endolymphatic sac and often lies on top of the sigmoid sinus (SS) behind the subarcuate fossa (FS) *Heidenhain-Susa* × 23

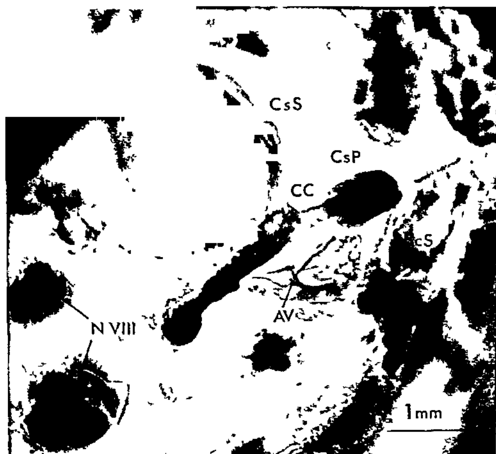



Fig 3 Macroscopic view of the posterior surface of a temporal bone The bone is drilled away over the posterior (CsP) and superior (CsS) semicircular canals and their common crus (CC) The medial wall of the posterior part of the vestibular aqueduct (AV) has been taken away and the blood vessels in the connective tissue surrounding the intraosseus part of the endolymphatic sac are visible as well as the sigmoid sulcus (ScS) close to the external aperture of the vestibular aqueduct The subarcuate fossa (FS) and the pores where the eight nerve enters the bone (N VIII) are clearly visible Temporal bone preparation (right side) × 20

As a background for this investigation, it seems justifiable to here give a short description of the normal anatomy of the vestibular aqueduct endolymphatic duct and sac in the guinea pig

## *Vestibular Aqueduct*

The osseous vestibular aqueduct begins as a slit like opening in the medial wall of the vestibule where it is separated from the superior sinus of the utricle by a thin bony lamella which constitutes the lateral wall of the aqueduct

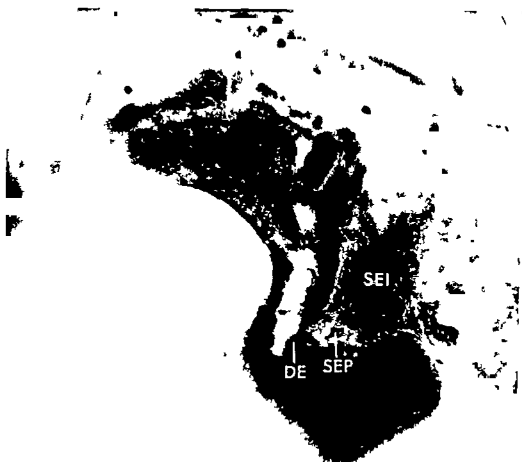



Fig 4 The same preparation as fig 3 The medial wall of the vestibular aqueduct is drilled away and the whole endolymphatic sac is visualized DE endolymphatic duct SEP proximal portion SEI intermediate portion and SED distal portion of the endolymphatic sac SS sigmoid sinus Temporal bone preparation (right side) × 20

It continues in a superior and posterior direction and penetrates through the bone in a slight curve medially and parallel to the common crus of the superior and posterior semicircular canals

In close proximity to the posterior surface of the pyramid it gradually widens follows the medial margin of the subarcuate fossa and finally ends as a 1 mm long narrow opening close to the sigmoid sulcus (Fig 2 and 3 The total length of the osseous vestibular aqueduct is approximately 2 5 3 mm in the adult guinea pig

## *Endolymphatic Duct*

The endolymphatic duct extends from the junction of the saccular and utricular ducts at the medial wall of the vestibule and ends half way down the vestibular aqueduct

It begins as an irregularly dilated sinus at the junction of the utricular and saccular ducts but as it enters the vestibular aqueduct, it narrows to form a constricted tube, the isthmus, which after approximately 1 5 mm again widens to become the first part of the endolymphatic sac (Fig 2 and 4)

The epithelial lining is squamous and separated from the endosteum of the bony aqueduct by a loose connective tissue The mucosa has a somewhat wavy appearance in the longitudinal direction but no crypts or protruding papillae are seen

## *Endolymphatic Sac*

In the posterior half of the vestibular aqueduct the endolymphatic duct dilates to a flat funnel shaped appendage called the endolymphatic sac This can be described as being divided into three parts (Fig 2 and 4)

*The proximal portion* is the first dilatation of the sac, which is completely located inside the vestibular aqueduct, and constitutes the transition between the endo lymphatic duct and sac proper It is approximately 0 5 mm long

The epithelium here is cuboidal and shows a few crypts and intraluminal projections The connective tissue is more vascular than that of the duct

*The intermediate portion* or the main part of the sac, is funnel shaped and lies partly within and partly outside the vestibular aqueduct and has an epithelial lining of considerably varying size and shape The length is about 2 mm and it is approximately 1 mm wide at the external orifice of the vestibular aqueduct

The epithelial cells range from cuboidal to high cylindrical in type and the walls are ruffled with many crypts and intraluminal papillae

The connective tissue is of the loose areolar type A dense network of thin walled capillaries lies just under the epithelium

The endosteum of the surrounding bone and, in the part outside the vestibular aqueduct the dura are closely related to the connective tissue of the sac

*The distal portion* is somewhat flattened and lies in close contact with the sigmoid sinus Its length is variable depending on how much the sac projects over the sigmoid sinus, but is usually about 0 5 mm in the adult guinea pig It has a very narrow lumen without crypts or protrusions

The epithelial lining changes gradually from cylindrical to low cuboidal cells

The connective tissue blends with that of both the dura and sigmoid sinus

## *Vascular Supply of the Endolymphatic Duct and Sac*

The endolymphatic duct is partly supplied with blood from a branch of the internal auditory artery Most of the blood to this system, however, is derived from a branch of the posterior meningeal artery, which penetrates the dura and connective tissue around the endolymphatic sac and forms a plexus over the distal widening part of the vestibular aqueduct (Bast and Anson, 1949)

The vein of the vestibular aqueduct receives blood from part of the utricle and semicircular ducts It anastomoses with the venous plexus of the endolymphatic sac and finally drains into the sigmoid sinus (Fig 3)

# Material and Methods

## *Animals*

Throughout this study young adult guinea pigs with a normal Preyer reflex, without signs of otitis media, and with a body weight of 250 to 350 grams were used. Specimens were obtained from approximately 25 guinea pigs for routine anatomical and histological studies and those from a further 20 animals were examined with the electron microscope to study the normal ultrastructure of the endolymphatic duct and sac.

## *Fixation*

For satisfactory preservation of the membranous labyrinth the fixative must be injected into the blood stream (Werner, 1936) or into the endolymph or perilymph whilst the animal is still alive or within the first minutes after death. In a study by Wersall, Kimura and Lundquist (1965) postmortal changes in the organ of Corti in guinea pigs were described as early as 10 to 15 minutes after death, and therefore all specimens used in this investigation were fixed not later than 5 minutes after the animal's execution.

*Fixation technique for light microscopy*

The animal was anaesthetized with Nembutal, the thorax opened and the heart exposed. A small glass cannula was introduced into the ascending aorta through the left ventricle and the animal was perfused with cold Ringer solution followed by Heidenhain Susa as the fixative. In order to facilitate the perfusion, the right side of the heart was widely opened so the blood could escape more easily. In most animals the abdominal aorta was clamped so that the fixative was concentrated to the upper part of the body.

A satisfactory fixation was indicated by the immediate de colouring and stiffening of the animal. After perfusion with approximately 0.5 liter of the fixative, the animal was decapitated, the bulla tympanica was opened on both sides and the head was immersed in the fixative and prepared for subsequent embedding in celloidin.

*Fixation technique for electron microscopy*

I I investigation all the specimens were fixed with 1 % osmium tetroxide solu
d with veronal acetate to pH 7.2—7.4 (Rhodin, 1954) and cooled with
r cess of fixation. The animals were killed by decapitation, the head
d the cranium opened after which the brain and cranial nerves
n disturbing the endolymphatic sac and its adherence to

the sigmoid sinus With a fine knife the sinus was opened, the blood clot removed and the osmium solution introduced into the sinus and on that part of the endolymphatic sac lying outside the vestibular aqueduct (Fig 4)

The temporal bone with the intact sac was dissected out and the bulla tympanica was widely opened with bone forceps Using a very fine knife the round window was opened and a small hole was made in the apical turn of the cochlea After breaking the small bony bar which passes between the crura of the stapes, the latter was pushed down into the vestibule itself

The fixative was now introduced both into the vestibule through the foramen ovale and into the apex of the cochlea through the hole already described This maneuver was facilitated by using a fine glass pipette Next, the entire specimen was immersed in ice cold fixative for three hours and finally, as will be described later it was embedded in Epon epoxy resin

## Preparation

*Preparation for light microscopy*

The entire skull, after perfusion with Heidenhain Susa solution, was decalcified in a solution of 5 % trichloroacetic acid and dehydrated by using increasing concentrations of ethanol before being finally embedded in celloidin

15 $\mu$ thick sections were cut on a Leitz celloidin microtome and the specimens were oriented with the modiolus of the cochleae in the same horisontal plane and parallel to the knife Every fifth section was stained with haematoxylin and eosin and mounted in canada balsam

Microscopy and photography were performed with a Zeiss Photo microscope

For more detailed cytological studies 0 5—1 $\mu$ sections, cut with the Ultrotome and mounted in Epon, were used and studied either with phase contrast technique or normal light microscopy, stained with toluidine blue solution

*Preparation for electron microscopy*

After fixation in buffered osmium tetroxide for 3 hours, the specimens were placed in cold Ringer solution and the various parts of the endolymphatic duct and sac were dissected out 'under water

The intermediate and distal parts outside the vestibular aqueduct were generally prepared in one piece together with the adhering wall of the sinus To make this possible an incision was made transversely across the endolymphatic sac at the external aperture of the vestibular aqueduct and then the sac together with the adjoining dura was loosened from the bone

The proximal and intermediate parts of the sac inside the vestibular aqueduct were visualized after peeling off the thin bony wall of its medial side (Fig 4) Using a flat knife it was then possible with care to remove these parts from the bony canal The distal part of the endolymphatic duct could also be reached this way but generally it was removed from the vestibular side using a fine needle

In a few of the specimens the bone surrounding the sac and duct was ground away with a dental burr and the dura over the sac carefully stripped off It was thus possible by this method to prepare the entire endolymphatic sac together with the distal part of the endolymphatic duct in one piece

Subsequently the specimens were dehydrated using first ice cooled 70 % ethanol solution and then increasing the concentrations of this fluid at room temperature, before the specimens were finally embedded in Epon epoxy resin (Luft, 1961)

Thin sections of the Epon embedded tissues were cut with glass knives on a LKB Ultrotome, floated on diluted alcohole and picked up on copper grids coated with a thin formvar film (100 meshes per inch) Most of the sections were stained with uranyl acetate (Watson, 1958) and lead acetate (Karnovsky, 1961) Unless otherwise mentioned, all electron micrographs used to illustrate this work were made from sections stained both with uranyl acetate and lead acetate

All examinations were performed with a Siemens Elmiskop I electron microscope operated at 60—100 kV, using objective apertures of from 50 μ to 10 μ Exposures were made on Gevaert Litholine plates at primary magnifications ranging from 1,000 to 40,000 times

# Normal Ultrastructure of the Endolymphatic Duct

The normal ultrastructure of the endolymphatic duct and sac has only been described by the author, in conjunction with Kimura and Wersall (1964). This paper is a continuation of these earlier investigations and attempts to give a more detailed account of the normal ultrastructure of the endolymphatic duct and sac in the guinea pig

## Epithelial lining

The epithelium is of a simple squamous or low cuboidal type resting on a smooth basement membrane The height of the cells range from 2—4 μ with a length from 6—8 μ (Fig 5 and 6)

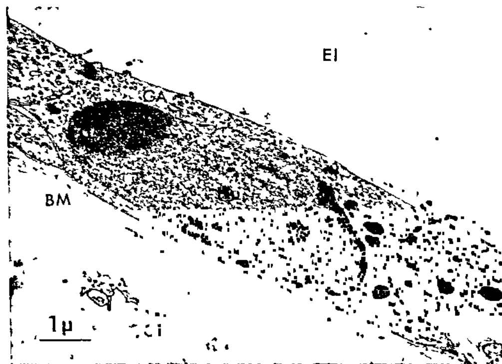



Fig 5 The squamous cells of the endolymphatic duct are smooth in the vestibular region The apical surface facing the endolymph (El) exhibits very few microvilli The cytoplasm has a few scattered mitochondria and sometimes an inconspicious Golgi apparatus (GA) can be found close to the nucleus (Nu) The basement membrane (BM) is thin and the connective tissue is of an areolar type (CT) × 13 000




Fig 6 The cells of the endolymphatic duct are more cuboidal in the distal part of the duct, with an irregular nucleus, often exhibiting one or more deep invaginations The basal part of the cells have sometimes one or more irregular protrusions giving the epithelial lining a wavy appearance (arrows) × 7,000

*Cytoplasm*

The cytoplasmic matrix appears dense with a few scattered ribosomes, similar to those described in other cell types (Palade, 1955) A centriole is sometimes seen

*Endoplasmic reticulum*

The cells of the endolymphatic duct contain short tubuli or membrane covered spaces evenly distributed in the cytoplasm These are covered with granules and represent the endoplasmic reticulum of the rough-surfaced type (Porter, 1961)

*Golgi apparatus*

Close to the nucleus in the peripheral part of the cell there is often found a collection of small vesicles scattered around vacuoles and cisternae similar to those described as the Golgi apparatus by Dalton and Felix (1956), but this structure is not very prominent in these duct type cells (Fig 6)

*Inclusion bodies*

Occasional lipid granules are seen together with a few small multi vesicular bodies Sometimes a few microbodies can also be found (Rhodin, 1954) These differing inclusion bodies are also found in the cells of the endolymphatic sac and will be more extensively described in connection with it

*Mitochondria*

Only a few mitochondria are found in the duct cells They are evenly distributed in the cytoplasm and are provided with the usual triple-layered outer and inner membranes (Palade 1952, Sjostrand, 1953) Generally, they are rounded with their internal membranes running transversely to the long axis They are 0 2—0 3 $\mu$ long

*Nucleus*

In the duct type cell, the nucleus is elongated with its greatest diameter parallel to the lumen of the duct and a size of about $2 \times 5$ $\mu$ It occupies a large portion of the cytoplasm and usually shows one or more deep invaginations of the nuclear membrane (Fig 6) A specific nucleolus is not present but the chromatin is condensed close to the nuclear membrane and its invaginations

*Cell borders*

The cell membrane consists of the usual triple layered unit membrane with two dense layers on either side of a less opaque one and a total width of about 100 A (Robertson 1959, 1961 Sjostrand and Elfvin, 1962)

The surface facing the lumen of the duct, bulges somewhat into it, due to the nucleus below and contains a few microvilli of maximum length of 0 2 $\mu$

The cell membranes between adjacent cells show the junctional complexes described by Farquhar and Palade (1963) with a tight junction closest to the lumen followed by a short intermediate junction and a desmosome Beneath the desmosome the membranes usually have a few interdigitating processes

The part of the cell membrane adjacent to the basement membrane is smooth In some cells one or more irregular protrusions of this basal part can be seen to project down into the subepithelial tissue for about 0 5 $\mu$ (Fig 6) These occur when a longitudinal wavyness is seen in the distal part of the duct

Subepithelial tissue

The basement membrane is continuous below the cells being about 0 1 $\mu$ thick and of a hyalin nature superficially but somewhat deeper it blends with the fibrils of the connective tissue The latter is of a very loose type containing a few collagen fibrils and fibroblasts

A few pigment cells are present in this subepithelial tissue in the vestibular part of the duct Capillaries are sparce and located at some distance from the epithelial lining




Fig 6 The cells of the endolymphatic duct are more cuboidal in the distal part of the duct, with an irregular nucleus, often exhibiting one or more deep invaginations The basal part of the cells have sometimes one or more irregular protrusions giving the epithelial lining a wavy appearance (arrows) × 7 000

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*Mitochondria*

Only a few mitochondria are found in the duct cells They are evenly distributed in the cytoplasm and are provided with the usual triple-layered outer and inner membranes (Palade, 1952, Sjostrand, 1953) Generally, they are rounded with their internal membranes running transversely to the long axis They are 0 2—0 3 μ long

*Nucleus*

In the duct-type cell, the nucleus is elongated with its greatest diameter parallel to the lumen of the duct and a size of about 2 × 5 μ It occupies a large portion of the cytoplasm and usually shows one or more deep invaginations of the nuclear membrane (Fig 6) A specific nucleolus is not present but the chromatin is condensed close to the nuclear membrane and its invaginations

*Cell borders*

The cell membrane consists of the usual triple-layered unit membrane with two dense layers on either side of a less opaque one and a total width of about 100 Å (Robertson, 1959, 1961, Sjostrand and Elfvin, 1962)

The surface facing the lumen of the duct, bulges somewhat into it, due to the nucleus below and contains a few microvilli, of maximum length of 0 2 μ

The cell membranes between adjacent cells show the junctional complexes described by Farquhar and Palade (1963) with a tight junction closest to the lumen followed by a short intermediate junction and a desmosome Beneath the desmosome the membranes usually have a few interdigitating processes

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Subepithelial tissue

The basement membrane is continuous below the cells, being about 0 1 μ thick and of a hyalin nature superficially but somewhat deeper it blends with the fibrils of the connective tissue The latter is of a very loose type containing a few collagen fibrils and fibroblasts

A few pigment cells are present in this subepithelial tissue in the vestibular part of the duct Capillaries are sparce and located at some distance from the epithelial lining



Fig 9 Detail of fig 7 showing the Golgi apparatus (GA) visible in both cells, adjacent to the junctional complex at the intercellular border lines (arrows) Below this region a few interdigitating processes are visible × 23,000

*The inclusion bodies* are more numerous than in the endolymphatic duct although less than in the intermediate portion They consist mostly of lipid granules together with a few multivesicular bodies

*The mitochondria* are relatively few and evenly distributed throughout the cytoplasm They are either rounded or oval and have an approximate diameter of 0 3 μ

*The nucleus* has a general appearance similar to that in the duct and here also shows deep invaginations However it is somewhat more rounded, is 3—4 μ in diameter and located in the middle of the cell (Fig 9) The chromatin is condensed around the nuclear membrane A nucleolus is not present

*The cell borders* The apical part of the cell membrane is convex and has more microvilli than in the endolymphatic duct Here these are also short (about 0 2 μ) and sometimes small pinocytotic vesicles can be found at their base

The side walls which are higher than in the duct exhibit a tight junction closest to the lumen, followed by a short intermediate junction and one or more desmosomes The main part of the side walls show interdigitating processes from neighbouring cells (Fig 8 and 9)

The basal cell border is more undulated than in the duct and small vesicles can sometimes be found close to it (Fig 10)

Fig 10 The irregular nucleus with condensed nucleoplasm at the periphery and the wavyness of the basal part of the cell is demonstrated A few small vesicles can be found in this area Detail of fig 7 × 18,000




Fig 11 Cells containing pigment like inclusions, are occasionally found in the subepithelial tissue of the endolymphatic duct and sac They are mainly seen in the intermediate portion, although this cell is from the proximal portion × 17,000

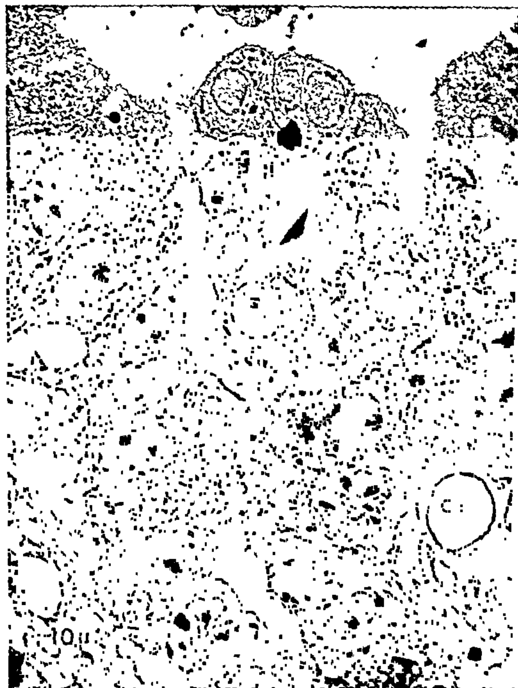



Fig 12 The intermediate portion of the endolymphatic sac is characterized by an epithelial lining with crypts and protruding papillae into the lumen of the sac Two types of cells can be distinguished, Light cells with a rounded nucleus and a "light" cytoplasm (LC), Dark cells having an irregular nucleus a dense cytoplasm and often a wedge shaped appearance (DC) Capillaries are present close to the epithelial lining (Ca) × 2,000




Fig 13 A light cell (LC) from the intermediate portion of the endolymphatic sac with a smooth rounded nucleus and a cytoplasm containing many vacuoles and vesicles. The apical surface has many microvilli and the basal part of the cell is smooth The adjacent dark cells (DC) have a more dense cytoplasm × 9 000

## Subepithelial tissue

The subepithelial tissue is more vascularized than in the duct, the capillaries lying close to the epithelial lining These occasionally exhibit the pores in their endothelial lining which will be described in the intermediate part Collagen fibrils are not so

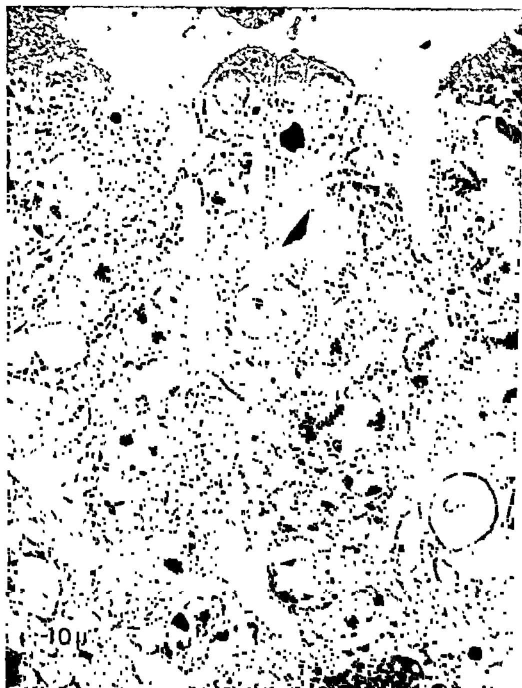



Fig 12 The intermediate portion of the endolymphatic sac is characterized by an epithelial lining with crypts and protruding papillae into the lumen of the sac Two types of cells can be distinguished, Light cells with a rounded nucleus and a "light" cytoplasm (LC), Dark cells having an irregular nucleus a dense cytoplasm and often a wedge shaped appearance (DC) Capillaries are present close to the epithelial lining (Ca) × 2,000

## *Intermediate Portion*

The intermediate portion begins within the vestibular aqueduct and constitutes most of the wider part, as well as most of the somewhat crescentic part outside the vestibular aqueduct

It has an epithelial lining consisting of cylindrical cells, irregularly arranged in protruding papillae and crypts (Fig 12) The height of the cells vary from 10—20 μ depending on their location, and they have a width of 4—8 μ

Two different types of cells can be distinguished, the light cells and the dark cells, which differ in their general shape and fine structure (Lundquist *et al*, 1964)

The light cells are more regular with a rounded nucleus and many microvilli at their luminal surface (Fig 13) The dark cells have a more condensed cytoplasm, an elongated nucleus and are often wedge shaped with a narrower basal part (Fig 14)

### Epithelial lining

*Cytoplasm*

In the *light cells,* the cytoplasm appears to be composed of a homogenous relatively pale matrix which, however, is slightly denser in the nuclear region and occasionally contains very fine fibrils

Ribosomes are richly abundant and mostly arranged in small clusters in the apical and basal parts of the cell

In the *dark cells,* the cytoplasmic matrix is very dense and has a fibrillous appearance Ribosomes although less numerous than in the light cells are more common than in the cells of the proximal part of the sac

*Endoplasmic reticulum*

In the *light cells* the so-called rough surfaced reticulum consisting of sheaths of thin paired membranes enclosing a narrow central space is most commonly seen On the cytoplasmic side, these membranes are covered with small ribosomes or ribonucleoprotein particles (Porter, 1961) The rough surfaced endoplasmic reticulum is richly abundant mostly in the supranuclear part of the cell close to the Golgi zone

The *dark cells* are not so richly provided with endoplasmic reticulum but where it is present it is usually of the rough surfaced variety (Fig 15)

*Golgi apparatus*

In the *light cells,* the Golgi complex is often prominent and located in the supranuclear region of the cytoplasm It consists of a system of paired smooth-surfaced membranes bounding thin slit like spaces which often dilates to form vacuoles of varying sizes These have a whole spectrum of densities from clear on the one hand to semi-opaque on the other Encircling this area, is a vesicle studded cytoplasmic matrix which is slightly more dense than the surrounding cytoplasm

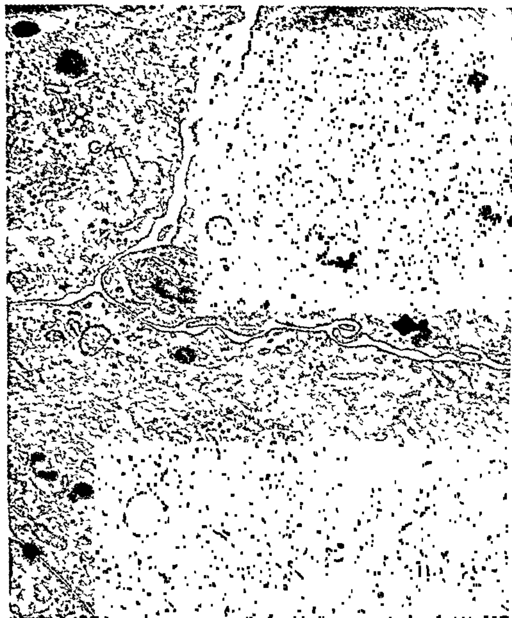



Fig 15 Tangentially cut cells from the intermediate portion showing the dense fibrillar appearance of the cytoplasm of the dark cells Endoplasmic reticulum and ribosomes are scattered around the Golgi apparatus (GA) × 24 000

The vacuoles range in size from 0 1 to 0 5 μ and the small vesicles have an approximate diameter of 500 Å

Sometimes a centriole is found in close relationship to the Golgi zone (Fig 16)

In the *dark cells* the Golgi apparatus is not so orderly arranged, but is seen as a vacuolar region in the supranuclear part of the cytoplasm (Fig 15)

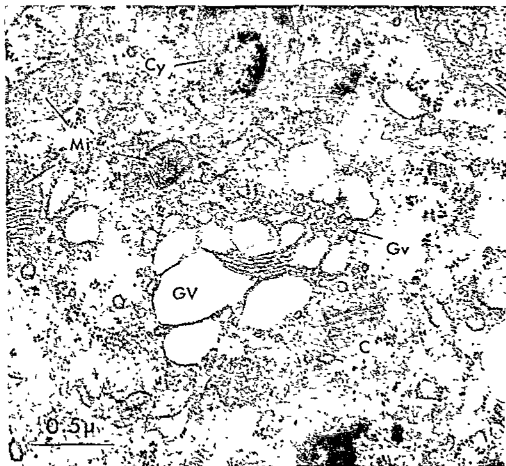



Fig 16 Golgi apparatus in a light cell showing the thin membrane bounded spaces, the Golgi vesicles (Gv) and vacuoles (GV) and the denser cytoplasm of this area A centriole (C) is found close to the Golgi zone as some mitochondria (Mi) and a unit membrane bounded cytosome like inclusion (Cy) × 42,000

*Inclusion bodies*

In this part of the sac and especially in the light cells, many cellular inclusions can be found Whereas all types of inclusion bodies are found in both the light and dark cells, they are more frequent in the light cells.

*Multivesicular bodies* (cf Rhodin 1963) are limited by a single membrane, have an approximate diameter of 0 5 μ, and contain many small vesicles The matrix of the multivesicular body is moderately dense Generally they are not very common, but one or two of these inclusions can be found in the apical and basal parts of the cytoplasm of the light cells (cf Fig 8).

*Microbodies* have been described in the kidney by Rhodin (1954) as small oval bodies surrounded by a single membrane and with a moderately stained matrix.

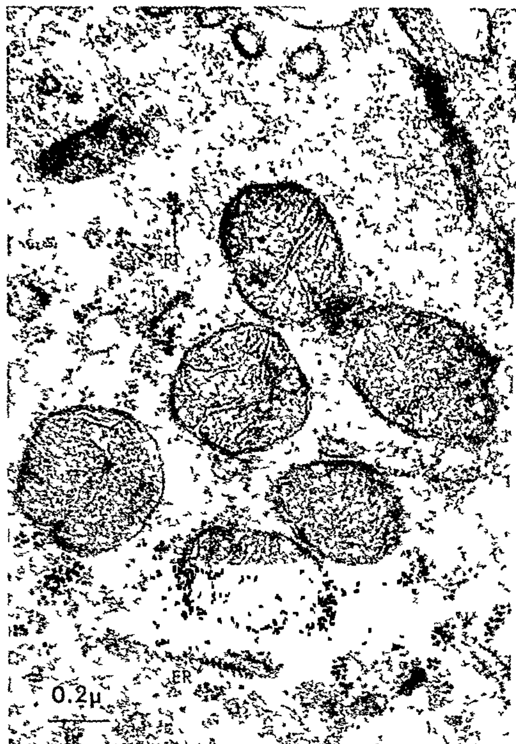



Fig. 17. The mitochondria in the intermediate portion are mostly oval shaped with their internal membranes running in a transverse direction to the long axis. Rough surfaced endoplasmic reticulum (ER) and small clusters of ribosomes (Ri) are often found in the near vicinity of the mitochondria. × 74 000

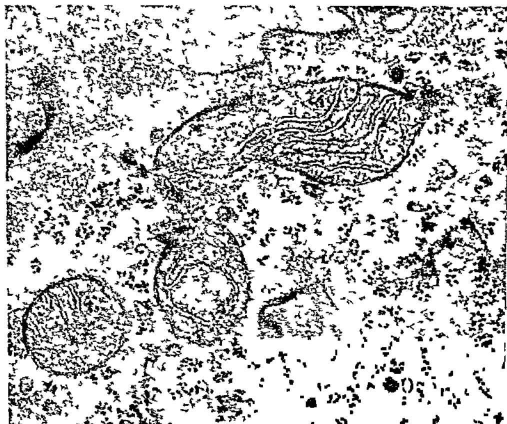


Fig 18 Mitochondria can be found where the internal membranes run more obliquely Small microbody like inclusions can also be seen among the mitochondria (arrow) × 56 000

Their size is 0 1—0 3 μ Cytoplasmic inclusions similar to this description are present in the intermediate part of the endolymphatic sac and are seen in the apical cytoplasm of predominantly the light cells

*Cytosomes* These inclusion bodies are limited by a single membrane of unit membrane type (Ericsson 1964) vary in diameter from 0 3—1 0 μ and contain large amounts of dense filamentous material They are however mostly to be seen in the normal endolymphatic sac as round bodies of 0 5 μ in diameter filled with fine granular material (Fig 16)

*Lipid granules* with high electron density and irregular outline are present in both light and dark cells In a specimen taken from an elderly guinea pig lipid granules together with pigment like granules were seen more commonly than in the younger adult animal (Lundquist *et al* 1964)

*Mitochondria*

Although both cell types of the intermediate part are rich in mitochondria they predominate in the light cells



Fig 19 Detail of nuclei from a dark and light cell respectively The nucleus of the dark cell (left) is irregular and very dense compared with the smooth nucleus of the light cell (right) The respectively dark and light cytoplasm is also visible × 22,000

These mitochondria are oval or rod-shaped with a smallest diameter of about 0 3 μ and a length of about 0 7 μ They exhibit the usual triple-layered outer and inner membranes as described in many other cells such as those of the kidney (Sjöstrand, 1953, Rhodin, 1954) the inner ear (Wersall, 1956) and the muscle (Andersson-Cedergren, 1959) These inner membranes mostly go in a direction transverse to the long axis of the mithochondria (Fig 17) but they occasionally can run in the long axis itself (Fig 18)

*Nucleus — light cells*

The nucleus is 5—7 μ in diameter and has a smooth nuclear membrane with the outermost layer showing a slight wavyness It is round in shape and located in the




Fig 20 Apical surface of a light cell exhibiting numerous microvilli and pinocytotic vesicles × 34,000

middle of the cell with a nucleoplasm less dense than that in the dark cell (Fig 19) Condensation of nucleoplasm along the nuclear envelope only occurs in one or two places but a nucleolus with high density is often found

*Nucleus — dark cells*

The nucleus almost fills the cell and has a height of up to 10 μ and a width of about 3 μ It is very elongated and often irregular with invaginations and condensation of nucleoplasm at the nuclear membrane and its invaginations

*Cell borders*

The apical surface is provided with numerous microvilli, being most pronounced in active light cells (Fig 20) These microvilli have a diameter of approximately 0 1 μ and a length of up to 0 6 μ (Fig 21) In close relation to them are seen invaginations of the apical cytoplasm and both small vesicles of about 0 1 μ in diameter and larger vacuoles of about 1 μ in diameter, all of which indicate a pinocytotic activity The endolymph in these regions seems to be more opaque and very fine electron dense granular material is often found around the villiform



Fig 21. High resolution picture of a microvillus where the triple layered unit membrane is clearly visible (arrows) A small pinocytotic vesicle is also found in the cytoplasm (Pv). The endolymph close to this area is granular and appears condensed × 180,000

protrusions (Fig 21) A primitive cilium has, a few times, been seen originating from a centriole at the fluid surface. A basal foot as described by Flock (1965) can be found at the basal body of the cilium (Fig. 22).

The neighbouring cell membranes are interconnected with junctional complexes (Fig. 23) as described by Farquhar and Palade (1963).

The apical part of this complex is the *zonula occludens* (tight junction) which is characterized by partial fusion of the adjacent cell membranes resulting in a local obliteration of the intercellular space closest to the lumen, which sometimes give the fused unit membranes a five layered appearance in a similar manner to that




Fig 22 A primitive cilium is sometimes seen originating from a centriole at the fluid surface and which protrudes into the endolymph A basal foot (arrow) is found attached to the basal body A centriole cut lengthwise is seen in the adjoining cell (C) × 36,000

described in the myelin lamella (Robertson, 1961) or in the retina (Nilsson, 1964) The cytoplasm is diffusely condensed in this region which is up to 0 4 μ long (Fig 24)

The cell membranes below this region diverge somewhat but close again to form the *zonula adhaerens* (intermediate junction) in which the cell walls are parallel with a narrow intercellular space of about 200 Å The cytoplasm closest to this region is diffusely dense The length of this part of the junctional complex varies from 0 2—0 5 μ (Fig 24)

The last part of the junctional complex according to Farquhar and Palade (1963) is the *macula adhaerens* (desmosome) which is noticed further down This is characterized by two oval plaques of dense material in the cytoplasm close to the inner layer of the plasma membrane from which it is separated by a small space of about 40 Å The inner layer of the plasma membrane is slightly denser than normally and can together with the outer dark part of the same membrane, be followed through the desmosome The space between the plasma membranes is around 250 Å and is occupied by a central dense membrane with the same length as the cytoplasmic plaques that is around 0 3 μ (Fig 25)

Below these junctional complexes the cell membranes interdigitate freely with one another this being more pronounced towards the base of the cells




Fig 23 Junctional complexes are fully developed in the cells of the intermediate portion (JC) This part of the adjacent cell membranes is terminated by one or more desmosomes Below this zone the cells show several interdigitations. × 20 000

In the *light cells* the base is generally smooth but even here characteristic invaginations can be found which are often irregular with fingerlike interdigitations as between neighbouring cells although the space between the basal processes is narrower Small vesicles are often scattered around this area (Fig 26) These basal invaginations are most developed and most easily found in active light cells The basement membrane is smooth and does not project into these invaginations




Fig 24 The junctional complexes consist of a *zonula occludens* close to the lumen The outer layers of the adjacent plasma membranes fuse in this region (ZO) Below follows a zone with parallel cell membranes and a slightly condensed cytoplasm the *zonula adhaerens* (ZA) The third part is the desmosome (D) with its cytoplasmic dense plaques and dense intermediate line × 90 000

The *dark cells* are more irregular often wedge shaped and show a wrinkled basal portion where small cytoplasmic papillae extend into the subepithelial tissue for 1–2 μ The basement membrane follows these papillae closely (Fig 14)

Fig 25 The desmosome consists of dark cytoplasmic plaques very close to the innermost layer of the plasma membrane which is more dense than usual Between the adjacent outer layers of the plasma membrane there is found an extra dark intermediate line (arrow) These three dark membranes have the same intensity × 300 000

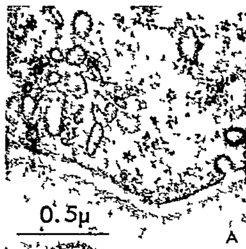






Fig 26 Rows of small vesicles are often found in the basal part of the light cells (A) These vesicles are mostly seen together with some interdigitations of the basal cytoplasm (B) The basement membrane does not enter the space between these small finger like processes × 45 000 (A) and × 30 000 (B)

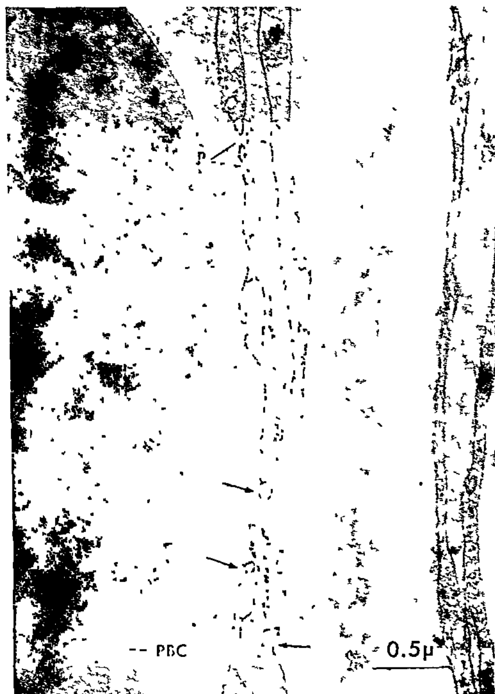



Fig 27 Detail of the endothelial wall of a capillary in the intermediate portion of the sac A red blood cell (RBC) is seen to the left close to the capillary endothelium where several small vesicles (arrows) and pores (P) are found × 40 000




Fig 28 A Detail of the pore in fig 27 A thin dense diaphragm covers the pore In the centre of the diaphragm a small dense 'knob' (arrow) is often found clearly visible in the detail of a second pore from the same region (B) × 350,000 (A and B)

### Subepithelial tissue

In the intermediate portion of the sac, the connective tissue is of areolar type with a rich capillary network in close relationship to the epithelial lining (Fig 12) These capillaries very often exhibit pores in their endothelial lining (Fig 27) similar to those described in the kidney (Rhodin 1962) These pores are about 550 Å in diameter and are covered by a thin membrane which often has a small dense "knob" in the centre (Fig 28)

The loose connective tissue has only a few collagen fibrils but spindle shaped fibroblasts and histiocytes are numerous Wandering leucocytes are also present and can sometimes be seen penetrating in between the epithelial cells of the endolymphatic sac

Further away from the epithelial lining the connective tissue is more fibrous and blends with the endosteum of the surrounding bone or with the dura in that part outside the vestibular aqueduct

## *Distal Portion*

This is the flattened terminal part of the sac which often lies partly on top of the sigmoid sinus

### Epithelial lining

The epithelial lining is cuboidal except at the extreme end where it is squamous It has a height of up to 10 $\mu$ and a width of about 5 $\mu$ The cells have the same appearance as those of the intermediate portion and both *light* and *dark cells*

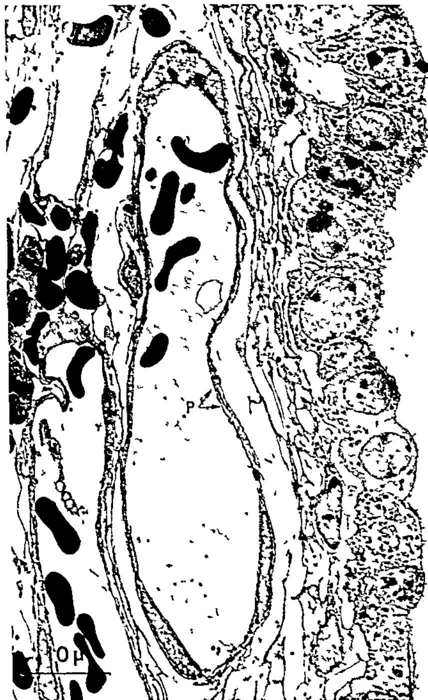



Fig 29 Survey picture of the distal portion of the endolymphatic sac In the upper part of the picture the cells are like those of the intermediate portion Further down they are more cuboidal in shape and with less microvilli on their apical border The connective tissue is loose and richly vascularized Capillaries can be found close to the epithelial lining These capillaries often exhibit pores (P) as described in the intermediate portion × 2 000



Fig 30 Free floating cells close to the epithelial lining of the intermediate portion of the endolymphatic sac These free floating cells are rounded and contain several vacuoles and other inclusions Their cytoplasmic borders are often provided with numerous filiform protrusions In the lower right hand corner of the picture, a degenerating cell from the epithelial lining can be seen together with a light and dark cell to the left × 5,000

can be seen In this part also, the light cells predominate These cells exhibit similar characteristics to those of the intermediate portion but seem to be much less active Their luminal surface is smooth with a few microvilli and the basal part of the cells is also smooth with a straight basement membrane underneath (Fig 29)

In the extreme end of this portion the cells are identical with the duct-type cells and are very closely related to the surrounding fibroblasts

## Subepithelial tissue

The connective tissue of the distal portion is richly vascularized with a network of capillaries which exhibit pores in their endothelial lining, as in the intermediate part (Fig 29) In that part adjacent to the sigmoid sinus this connective tissue completely blends with that around the sinus

## *Normal Contents of the Lumen of the Endolymphatic Sac*

The lumen of the sac is very irregular in the intermediate portion due to epithelial papillae which sometimes almost cross the entire lumen These papillae are often separated by narrow crypts where the endolymph seems to be condensed and granular in appearance

Close to the epithelial lining cellular debris is found consisting partly of de generating epithelial cells and partly of degenerating free floating cells (Guild 1927 Fig 30)

### Free floating cells

The endolymphatic sac is normally provided with a population of free floating cells predominantly rounded macrophages A few leucocytes are also present

The free floating macrophages are rounded with an irregular nucleus which shows a condensation of chromatin at the nuclear border Their cytoplasm is rela tively dense like that of the dark cells being filled with mitochondria and endo plasmic reticulum (Fig 31) Large vacuoles are often found filled with granular material These vacuoles sometimes fill the cytoplasm so as to give the cell a signet ring appearance (Guild 1927 Fig 32 and 33) The cytoplasmic border exhibits diffuse filiform protrusion as well as very prominent often filiform pseu dopodia with a cytoplasmic matrix without ribosomes (Fig 31)

Another type of these free floating cells has a very light cytoplasm filled with large dense inclusions which are regular in appearance and bounded by a thin mem brane (Fig 34 and 35) The plasma membrane is smooth and this cell does not exhibit any pseudopodia




Fig 31 Free floating cells from the intermediate portion of the sac They have a dense cytoplasm rich in endoplasmic reticulum and mitochondria. Many pseudopodia are found around the plasma membrane The nucleus is dense with chromatin condensed along the periphery In the cell in the lower left hand corner many large vacuoles are found together with pseudopodia with a less dense and diffuse cytoplasm (arrows) × 9 000

Fig 32 Details from free floating cell from fig 30 The cytoplasm is filled with large vacuoles which contain a granular substance The cytoplasm has a high concentration of ribosomes and a dark appearance The plasma membrane often exhibit diffuse filiform protrusions (f) × 17 000




Fig 33 This type of free floating cells contains so many large vacuoles as to have a "signet ring like appearance × 7 000

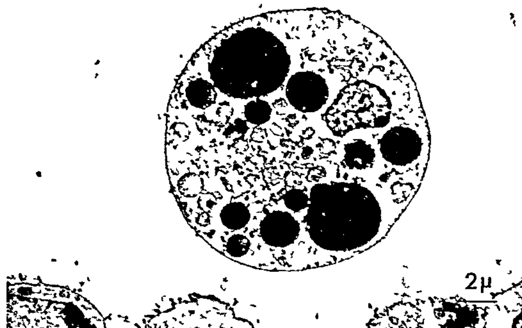



Fig 34 A free floating cell from the intermediate portion of the sac This type has a light cytoplasm containing several dark inclusions The plasma membrane is smooth without protrusions The apical part of dark cells of the epithelial lining can be found below the free floating cell × 5 000

Fig 35 Details from the free floating cell of fig 34 The very dark cytosome like inclusion bodies are bounded by a thin membrane (arrows) × 25 000

# Normal Ultrastructure of the Utricular and Saccular Ducts and the Sigmoid Sinus

A brief account will be given of the utricular and saccular ducts, which connect the endolymphatic duct to the utricle and saccule respectively As the distal portion of the endolymphatic sac partly protrudes on top of the sigmoid sinus and their connective tissue blands, a brief description of the fine structure of the sinus also seems to be of interest

## *Utricular and Saccular Ducts*

*The utricular duct* is narrow and begins obliquely at the utricle in a fold, the so-called utriculo-endolymphatic valve (Bast, 1928)
*The saccular duct* is a straight tube, oval in cross section (Fig 36)




Fig 36 The saccular duct, which connects the endolymphatic duct to the saccule, has a smooth cuboidal epithelium surrounding an oval shaped lumen The subepithelial tissue is areolar in type × 4 000




Fig 37 The epithelial cells of the saccular duct have a smooth appearance with cuboidal cells with a moderately dense cytoplasm containing a few mitochondria There are not found any inclusion bodies The surface of the cell has only a few microvilli The adjacent cell membranes are straight without interdigitations but the basal plasma membrane is wavy with a thin basement membrane below × 17 000

At their junction with the sinus of the endolymphatic duct the utricular and saccular ducts have a similar structure that is smooth cuboidal cells standing on a thin basement membrane and having an areolar subepithelial tissue

The epithelial cells have a height of 3—4 μ and a width of 2—3 μ and their cytoplasm is of medium density containing scattered ribosomes and vacuoles The endoplasmic reticulum is of the rough surfaced variety but not highly developed and a Golgi apparatus is present in the supra nuclear zone The mitochondria are few evenly distributed in the cytoplasm and of the oval or rod shaped type

The nucleus is slightly elliptical with an approximate diameter of 3 μ and sometimes shows invaginations of the nuclear border The nucleoplasm is moderately dense with chromatin condensed at the smooth nuclear membrane

The surface of the cell is smooth except for a few microvilli The side walls show the usual junctional complexes at their lumenal surface but below this region the neighbouring cell membranes lie parallel without any interdigitations (Fig 37) The base of the cell is undulated with a thin basement membrane which blends with the areolar tissue below in which as well as fibroblasts an occasional pigment cell can be seen

## *Sigmoid Sinus*

The distal portion of the endolymphatic sac often lies on top of the medial wall of the sigmoid sinus where they are in close relation to one another, with their connective tissue completely interlocking.

Below the vascularized zone of the connective tissue of the sac is a more fibrous zone, consisting of a thin layer of collagen fibrils and fibroblasts. However, as the endothelial lining of the sinus is approached, the connective tissue becomes of a loose areolar type again.

The endothelial lining of the sinus is extremely thin and consists of a single layer of characteristic cells which have an irregular, wrinkled appearance. They have an approximate length of 10 μ and a height of 0.5 μ except in the nuclear region where they are about 3 μ (Fig. 38 and 39).

The cytoplasm contains scattered ribosomes and many small pinocytotic vesicles, especially towards the lumen of the sinus and along the base. Close to the nucleus is the Golgi zone, and a centriole is often present in this region (Fig. 40). Inclusion bodies are absent except an occasional lipid granule but a few oval mitochondria are usually seen.

The nucleus is somewhat flattened with an extremely irregular outline due to small invaginations around the periphery and sometimes a small nucleolus can be seen often eccentrically located within the nucleoplasm. The nuclear membrane is also irregular with empty looking spaces between the nucleus and the cytoplasm.

The fluid surface shows a few microvilli over the nuclear region but is otherwise smooth. The neighbouring cell membranes partly overlap without any clear cut

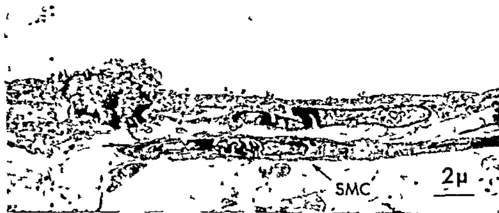



Fig. 38. The endothelial cells of the sigmoid sinus are thin and long with a nuclear zone slightly higher than the rest of the cell. Smooth muscle cells (SMC) are found beneath the endothelial cells. × 5 000.




Fig 39 The cytoplasm of the endothelial cells of the sigmoid sinus has a wrinkled appearance in the nuclear zones The nuclei are very irregular with wrinkled nuclear membranes The nucleoplasm is condensed along the periphery × 13,000

junctional complexes or interdigitations The basal cell border is smooth and many small vesicles are seen close to a granular basement membrane

Near the basal part of the cell are seen spindle-shaped smooth muscle cells which contain an elongated "spirally twisted" nucleus (Fig 38 and 40)

Deep to this zone is a loose meshwork of collagen fibrils related to the sub-epithelial tissue of the endolymphatic sac




Fig 40 Detail of a sinus cell with its Golgi apparatus (GA) and a centriole (C) close to the surface of the cell Immediately below a smooth muscle cell is found filled with myofilaments (arrows) × 22 000

# Discussion

The present investigation confirmed the accuracy of the description of the endo lymphatic duct and sac by earlier anatomists and histologists with regard to the cellular outline of the epithelial lining (Boettcher, 1869, Siebenmann, 1919, Iwata, 1924, Guild, 1927, Yamakawa, 1929, Siirala, 1942, Secretan, 1944 Bast and Anson, 1949 and others) They all agree that the part containing high cuboidal or cylindrical cells, the intermediate part (Guild, 1927), seems to be the most complex and is probably the most active part

They, however, differ in their opinions as to the possible functions of the endo lymphatic sac, as interpreted from a morphological study Thus in relating the structure to the possible function of the endolymphatic sac, the investigators have expressed the following views

1 The endolymphatic sac is a rudimentary organ probably without any specific function (Siebenmann, 1919)

2 Endolymph is produced by the sac (Boettcher, 1869, Hasse, 1881, Seymor, 1954 and 1960)

3 Endolymph is resorbed by the sac (Iwata, 1924, Guild 1927, Yamakawa, 1929, Siirala, 1942, Secretan, 1944, Andersen, 1948, Engstrom and Hjorth, 1950 and others) Many supporters of this view have observed intrasaccular cells with phago cytotic activity which might have importance in the maintenance of normal endo lymph

4 The function of the sac is to act as a pressure regulator for the inner ear either by reabsorbing endolymph or by being a passively distensible sac (Kolmer 1923, Anson and Bast, 1960, Allen, 1964)

Taking in consideration these views, the possible function of the various parts of the endolymphatic duct and sac will be discussed on a pure morphological basis

## *Endolymphatic Duct*

This part together with its connections with the saccule and utricle seems to be of very little physiological importance as judged by the morphology

The utricular and saccular ducts have an epithelial lining consisting of smooth cuboidal cells with few mitochondria and microvilli and sparse endoplasmic reti culum The general structure is similar to that of the epithelial lining on the side walls of the saccule and utricle (Smith, 1956)

The endolymphatic duct also has an epithelial lining consisting of low cuboidal cells These cells do not exhibit many signs of surface activity, so it seems likely that the endolymphatic duct is only a connection to the physiologically active part of the system, the endolymphatic sac

## *Endolymphatic Sac*

### Proximal portion

In the first dilated part of this system, within the widening part of the vestibular aqueduct, there is a zone of transitional epithelium, that is, an epithelial lining exhibiting duct like cells together with cells showing signs of surface activity There are more mitochondria and cellular inclusions than in the duct type cells This transitional zone constituting the proximal portion of the endolymphatic sac (Guild, 1927) gradually changes into a most complex and active part, the intermediate portion (Guild, 1927 Lundquist *et al*, 1964)

### Intermediate portion

The intermediate portion is easily identified by its high cylindrical cells which protrude into the lumen in irregular papillae or folds, sometimes almost transversing it Such an appearance can be seen both in the part of the sac which lies within the distal part of the vestibular aqueduct and in most of the part of the sac which lies intradurally (Lundquist *et al*, 1964) This confirms the work of Guild (1927) The proximal or rugose portion of the sac as considered by Bast and Anson (1949), is not a morphologically well defined part They named the whole part of the sac inside the vestibular aqueduct, the rugose portion This is both the proximal and the subosteal part of the intermediate portion as considered by Guild (1927) and the author of the present investigation This difference in opinion may be due to the fact that Guild's investigations were carried out on guinea pigs whereas Bast and Anson's were on human material Also electron microscopical technique makes it easier to accurately plot the different cell types

In the present investigation the light cells of the intermediate portion are seen to have many of the morphological criteria for cellular activity The surface of the cells facing the lumen of the sac, is richly provided with microvilli There is also a considerable pinocytotic activity indicated by the formation of small vesicles at the apical border, and the presence of many small pinocytotic vesicles and vacuoles in the apical cytoplasm

The Golgi apparatus, is more developed in the cells of the intermediate portion than in the rest of the sac and duct, and clearly demonstrates the various parts of the Golgi complex There are also found many mitochondria, an abundant endoplasmic reticulum and inclusion bodies of various kinds

The basal cell membrane is folded in a similar way as has been previously reported in cells specializing in fluid transport e g the kidney (Sjostrand and Rhodin 1953, Pease, 1955 and others), the choroid plexus (Maxwell and Pease, 1956) and the secretory cells of the ciliary body (Pease, 1956, Holmberg, 1957, Pappas *et al*, 1959) These infoldings form U-shaped loops or fingerlike interdigitations in the basal part of the light cells, often surrounded by a row of small vesicles as described in the ciliary epithelium (Holmberg, 1957) or in the high cylindrical cells of the gallbladder (Hayward, 1962)

The active fluid transport in this area is also indicated by the existence of the pores in the plasma membrane of the endothelial cells of the capillaries found in the subepithelial tissue Such pores or fenestrae have been described in capillaries in several organs important for fluid transport such as the kidney (Rhodin 1962) the choroid plexus and the ciliary processes (Pappas and Tennyson 1962)

The basement membrane is thin about 500 Å which explains why the earlier investigators have failed to notice this structure

The two types of cells present in the intermediate region seem to have different physiological activities The light cell with its numerous microvilli and rich vacuolar contents seems to be actively concerned with fluid reabsorption The function of the dark cell with its more fibrillar cytoplasm irregular nucleus and smoother apical border, is obviously not the same but is difficult to postulate from the morphological data and will be further discussed in the following experimental part

## Distal portion

The light cells are still seen in the distal part suggestive of a probable resorptive capacity as is also the areolar subepithelial tissue with its fenestrated capillaries as described previously Closer to the end of the sac the cells diminish in height and duct like cells are seen among the light and dark cells present At the extreme end only the duct type cells are found

These findings indicate that the distal part is the termination zone where the activity gradually diminishes and finally disappears The disappearance of the *crypts and protruding papillae and smoothening of the luminal surface of the* epithelial lining also suggest this In the terminal region the adjacent surfaces of the walls of the sac can be seen very close to one another but they fail to show any signs of activity

## Free floating cells

These cells appear first in the proximal portion but are most frequent in the intermediate part of the sac Their nature is doubtful though many of them can be identified as leucocytes Probably most of them are derived from the connective tissue and the dark epithelial cells They are often spherical in shape containing inclusion bodies and large vacuoles and they seem to exhibit both pinocytotic and phagocytotic activity as indicated by the experiments of several authors (Guild 1927 Katayama 1928 Andersen 1948 Engstrom and Hjorth 1950 Lundquist *et al* 1964)

Their submicroscopic structure shows many small pseudopodia and sometime a filamentous zone at the fluid side of the cytoplasmic border is seen similar to that described in the ameba during the pinocytotic uptake of ferritin (Brandt and Pappas 1962

EXPERIMENTS ON THE PHAGOCYTOTIC ACTIVITY OF THE ENDOLYMPHATIC SAC

# Introduction

The first thorough experimental investigation on the function of the endolymphatic sac was by Guild (1927) who injected potassium ferrocyanide into the cochlear duct and by adding hydrochloric acid during the process of fixation caused precipitation of Prussian blue granules in the parts of the labyrinth where potassium ferrocyanide was present Guild found that Prussian blue granules were accumulated in the endolymphatic sac and so he presented the "longitudinal flow" theory where endolymph is assumed to be produced by stria vascularis in the cochlear duct and from there flows via the saccule into the endolymphatic sac, where it is resorbed This view is confirmed by several other investigators who have performed intraperitoneal and intravenous injections of dyes and have been able to trace these to the endolymphatic sac and the perisaccular tissue, (Andersen, 1948 Engstrom and Hjorth, 1950) Other investigators, however, oppose the theory that the endolymphatic sac is essential for the reabsorbtion of endolymph, on the grounds that experimental obstruction of the endolymphatic duct and sac have failed to give any significant change in the morphology or function of the inner ear (Mc Nally 1926, Lindsay 1947 Schuknecht and Kimura 1953 Schuknecht and Seifi, 1963) It has even been suggested that the function of the endolympha tic sac is to secret endolymph (Boettcher, 1869, Hasse, 1881, Seymor, 1954)

These considerably differing views as to the function of the endolymphatic sac, have stimulated the author to perform a series of experiments designed to scrutinize, with the aid of electron microscopy the reaction in the endolymphatic sac to foreign particles and bacteria introduced into the endolymphatic system

## *Outline of Experiments*

The aim of this investigation was to study the endolymphatic sac after the labyrinth had been exposed to foreign material circulating in the endolymph In the first series of experiments, small particles such as ferritin, colloidal silver and gold were injected into the endolymphatic system through the lateral wall of the cochlear duct in guinea pigs These animals were at a later stage killed and their endolymphatic sacs studied

It was subsequently shown that silver particles were the most suitable for this type of work and therefore these were used throughout the investigations described in this paper The choice of particles will be further commented on in the discussion

In the second part of this experimental study living bacteria were introduced into the endolymphatic system in the same manner as the silver particles and the reaction of the endolymphatic sac to them was again studied

# Material and Methods

## *Experimental Animals*

Young adult guinea pigs with a normal Preyer reflex and a body weight of 250 to 350 grams were used. Non pigmented guinea pigs, albinos, were not used as the lack of pigment in their labyrinths make the surgical part of the experiment more difficult.

## *Injection Solutions*

**Silver solution**

An 0.25 per cent sterilized aqueous solution of colloidal silver was used (Fig. 41). This was prepared by The Central Military Pharmacy of the Swedish Defence Forces.

**Bacteria**

A group C hemolytic streptococcus (Fig. 42) was kindly provided by the Department of Bacteriology at Karolinska Institutet. This, together with a strain of E. coli used in one animal, was not pathogenic for man.

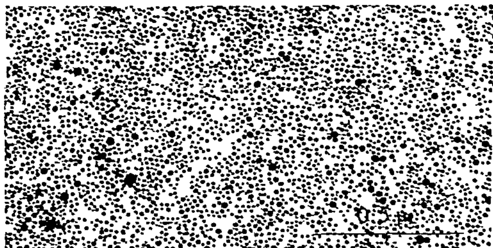


Fig. 41. The colloidal silver particles are of uniform size with a diameter of up to 250 Å. Unstained. × 75 000.

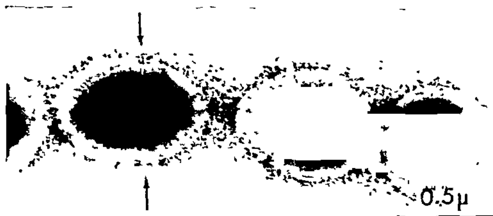



Fig 42 The bacteria often exhibit septae, indicating their zone of division (arrows) Group C hemolytic streptococcus. *Neg staining* × 47,000

Fig 43 The microinjection equipment consists of a microcapillary holder mounted on a micrometer which is attached to a Prior micromanipulator by a ball and socket joint

## *Microinjection Equipment*

Micro capillaries with an approximate tip diameter of 20—50 $\mu$ were used for the intracochlear injection experiments They were produced with the aid of a micro-electrode pulling machine from glass capillar es with an external diameter of 1 mm The capillaries were held by a modified Leitz capillary holder mounted on a micrometer which was attached to a Prior micromanipulator via a ball and socket joint (Fig 43)

The capillary was connected to a micrometer syringe with a polyethylene catheter It was thus possible to inject known quantities of fluid minimal 10 $\mu$l with an accuracy of one per cent

## *Surgical Technique*

The apical coils and most of the basal coils of the cochlea of the guinea pig protrude freely into the bulla tympanica of the middle ear cavity By removing the bone of the semispheral surrounding walls of the bulla it was possible to expose the various parts of the cochlea

In most cases an anterior approach through the neck and into the bulla tympanica was used but in a few cases a lateral approach with a retroauricular incision opening the middle ear cavity from a superior and lateral direction was preferred By using the latter method it was possible to expose the round window area

Work on the cochlea itself was performed under 40 times magnification using a Zeiss binocular operating microscope

### Anterior approach

The guinea pig was anaesthetized with a single intraperitoneal injection of Nembutal Abbot using 35 mg kg After shaving the operating field the animal was fastened in a head holder wi h the animal biting over a bar and with the maxilla and mandible squeezed together (Kimura 1963)

The animal was then turned on its back and fastened to the operating table A small amount of xylocain was injected subcutaneously in the wound area and a 20 mm long incision made in the neck parallel to the mandible

By careful dissection it was possible to retract the subcutaneous cervical artery and external jugular vein without ligating them The dissection was deepened towards the bulla tympanica by following the medial aspect of the mandible After having visualized the styloid process and dissected free the stylohyoid and styloglossal muscles a small specially made retractor was inserted between these and the other deep muscles of the neck in such a way that a good exposure of the bulla was obtained




Fig 44 A An anterior approach through the neck is most often used in the injection experiments The capillary tube can be followed into the opened bulla tympanica where the cochlea is clearly visible

An incision was made in the periosteum which could then be retracted and a series of small holes made into the bulla with an electrical drill The remaining bone was removed with fine bone forceps making an oblong hole 3×5 mm over the cochlea

*The cochlear duct could be identified inside the bone between the grooves on* the surface of the cochlea because of its vascularity and pigmentation

A small hole about 0 2 mm in diameter was made with a sharpened drill into the first and third coils of the cochlear duct The width of the external wall of the cochlear duct is about 0 4 mm in these regions A glass capillary 20—50 μ in tip diameter was introduced into the hole in the basal turn with the aid of a micro manipulator (Fig 44) and through this 50—100 μl of the experimental solution was slowly injected A satisfactory injection was indicated by the outflow of endo lymph from the hole in the third turn and the injection was continued until some of the experimental solution was noticeable from this hole Whereas the *brown colour of the colloidal silver solution was easily identified the bacterial solu*tion was not In the latter an injection of 100 μl was considered enough and the capillary was withdrawn




F g 44 B Schemat c drawing from f g 44 A llustra ng the nject on n the f rst turn of the cochlear duct A hole s also made r the th rd turn to perm t outflow of endolymph (arrow) × 23

The drill holes in the bony cochlear wall were carefully covered with sterile bone wax It was important not to pre s in the bone wax too hard so that it penetrated into the cochlear duct and blocked it The periosteum over the bulla was now replaced so that it covered the hole as did the stylohyoid and styloglossal muscles when the retractor was removed The skin incision was closed with a continuous locking suture

## Lateral approach

A retroauricular incision was made with the an mal in an abdominal position By freeing the insert ons of the sternocle domasto d and occipital muscles it was possible to visual ze the whole of the lateral bony wall of the middle ear right up to the emergence of the facial nerve and the auditory canal The wall was opened with

an electrical burr and the round window was seen and incised with a fine flat dissection needle, and part of the bony wall of the scala tympani was drilled away to give wider access to the basilar membrane A fine capillary with a tip diameter of about 20 μ, was pushed through the basilar membrane into the cochlear duct and a small amount of fluid was injected

Gel foam, washed in sterile saline solution was used to close the defect in the round window area and the wound was closed with a continuous locking suture

## *Fixation and Preparation for Electron Microscopy*

The animals were killed and the specimens were obtained and prepared according to the methods previously described (page 14)

The first sections were examined unstained in case the process of staining should render the silver particles unrecognizeable As it turned out these particles were very characteristic due to their uniform size (around 250 Å) and high electron density (Fig 41), it was therefore possible to employ the usual uranyl acetate and lead staining later, without loosing track of the silver particles These particles were also identified by selective area electron diffraction comparison in which the diffraction pattern of large intracellular inclusions was compared with that of colloidal silver granules isolated on a specimen grid

The bacteria were identified by their normal morphological characteristics and by cultivation

# Injection of Colloidal Silver Particles into the Cochlear Duct

Silver particles were injected into the cochlear duct of nine guinea pigs Seven using a hole in the first turn of the cochlea and two through the round window and through the basilar membrane of the first turn

The animals were killed and the inner ear on both the operated and control side, was removed, at the intervals 5 7, 20, 20, 24, 30 48, 48 and 48 hours after the injection

## General Findings

The injected guinea pigs did not exhibit any severe signs of intoxication or labyrinthitis, and behaved in a normal way when they recovered from the anaesthetic

### *Middle ear*

In the middle ear cavity no signs of inflammatory reactions with pus formation was seen and only a small amount of fibrin around the injection hole disturbed the normal anatomy

### *Cochlea*

The organ of Corti was destroyed in the area of injection Silver was, however found here as well as in the higher turns, where the organ of Corti was much less damaged and contained swollen but still recognizeable hair cells

Silver granules were also found below the basilar membrane sometimes inside monocyte-like cells

### *Vestibular apparatus*

The sensory cells of the vestibular part of the membranous labyrinth were much less damaged although some signs of degeneration were found They resembled hair cells in early stages of streptomycin intoxication with vacuoles in the cytoplasm and club like protrusions of the apical cytoplasm into the lumen (Duvall and Wersall, 1964) The underlying nerve endings were normal, silver granules not being observed in this region

No abnormalities were seen in the membranous labyrinth and endolymphatic sac from the uncontaminated side

The change in the cochlear duct and the vestibular apparatus caused by the silver granules will not be described further as the primary purpose of this investigation was to study the reaction of the endolymphatic sac to introduced foreign particles




Fig 45 The lumen of the endolymphatic sac is filled with electron dense silver particles (Ag) found partly inside the free floating macrophages and partly free in the fluid Cellular silver containing debris (arrows) neutrophil leucocytes (NLc) and red blood cells (RBC) are also often found in the lumen × 13 000

## *Findings in the Endolymphatic Sac*

Silver granules were found in the endolymphatic sacs of seven of the nine injected animals killed between 5 and 48 hours after the injection In the remaining two animals which had both been killed 48 hours after the injections silver granules were not found

The most pronounced activity in the endolymphatic sacs was demonstrated up to 24 hours after the injection of silver Thus all changes described could be found in the various parts of the intermediate portion of the sac of an animal killed at this time

### Free floating cells

The lumen of the sac was filled with free floating macrophages cell debris and silver particles (Fig 45)

The free floating cells were often of the rounded dark type (cf Fig 31) with many finger like protrusions at the periphery They contained many vacuoles




Fig 46 Dense free floating cells with small pseudopodia around the surface The cell on the left contains several inclusions filled with silver (Ag) and a big irregular inclusion of condensed appearance × 14 000

inclusion bodies consisting of dense material, and silver inclusions (Fig 46) The silver particles were mostly found in unit membrane covered inclusions in which the matrix had a condensed appearance sometimes resembling cytoplasmic residues

Wandering neutrophil leucocytes were also often seen These cells exhibited pseudopodia like projections which could be found reaching out and engulfing cellular debris containing colloidal particles (Fig 47) The cytoplasmic protrusions were dense with an amorphous matrix In the next stage cell debris was attached to the plasma membrane of the leucocyte and so engulfed into the cytoplasm (Fig 47) These various stages could often be demonstrated at the same time in the cell The cellular debris in the lumen of the sac consisted of dark irregular masses, often vacuolated and containing silver which could occasionally be identified as degener




Fig 47 The neutrophil leucocytes often exhibit pseudopodia reaching out towards cellular silver-containing debris This cell demonstrates several stages in the incorporation of the foreign material from pseudopodia formation (1) which when in contact with the debris (2) starts to encircle it (3) to the final engulfment of debris into the cytoplasm (4) Fine colloidal particles can be found in all these inclusions × 26 000

ating macrophages epithelial cells and red blood cells (Fig 45) Some of these cells were extremely vacuolar and had the signet ring like appearance described by Guild (1927)

## Epithelial lining

The epithelial cells of the intermediate portion were more active than normally especially in the crypts of this region where cellular debris free floating cells and silver had accumulated The endolymph seemed to be condensed in the crypts giving their fluid space a finely granular appearance (Fig 48)




Fig 48 The light cells exhibit a marked increase in pinocytotic activity with long microvilli and pinocytotic vesicles at their fluid surface The endolymphatic space has a granular condensed appearance in these regions × 48,000

*The light cells*

The light cells exhibited marked pinocytotic activity with long villiform protrusions and many pinocytotic vesicles in their apical cytoplasm (Fig 48)

Invaginations into the cytoplasm were sometimes deep with only a narrow connection to the cytoplasmic border and contained finely granular material (Fig 49) A thin membrane could sometimes be seen to close off this invagination (Fig 50) Larger pinocytotic vacuoles deeper in the light cells, with an appearance similar to those described in the earlier part of this work were also noticeable (cf page 33) Sometimes small vesicles and vacuoles containing silver were demonstrated

In the basal region, the finger-like invaginations of the cell border were more prominent than is normally the case and will be more fully described later in this paper (page 88)

*The dark cells*

The dark cells showed a striking change with a somewhat less condensed appearance of the cytoplasm and marked macrophage activity




Fig 19 Small invaginations were often found at the apical surface of the epithelial cells The contents of these invaginations are often granular in appearance (arrow) The three layers of the plasma membrane can be clearly seen × 140 000

Free silver particles were found together with fine granular material, condensed at the fluid surface of these cells (Fig 51) and surrounding these silver particles finger like projections of the apical cytoplasm were seen The plasma membrane appeared irregular with diffuse areas where the cytoplasmic border could not be followed (Fig 52) In some specimens, especially in the bottom of the crypts, protrusions from neighbouring cells interdigitated so as to enclose the silver particles (Fig 53)

Inclusions filled with silver particles and bounded by a unit membrane were found in the apical cytoplasm (Fig 54) as well as in the basal part of the cells (Fig 55) Silver granules were always found as cytoplasmic inclusions and never in the intercellular space

Not only were silver granules taken up by these dark epithelial "macrophage cells, but even red blood cells were observed apparently in the process of being phagocytized (Fig 56)

Degenerating cellular debris of unidentifiable origin was often found in close contact with the surface membrane of these dark cells for a distance up to more than 1 μ and surrounded by protrusions from the apical cytoplasm (Fig 57)

Degenerating epithelial cells which seemed to have been shed into the lumen of the sac could also be found

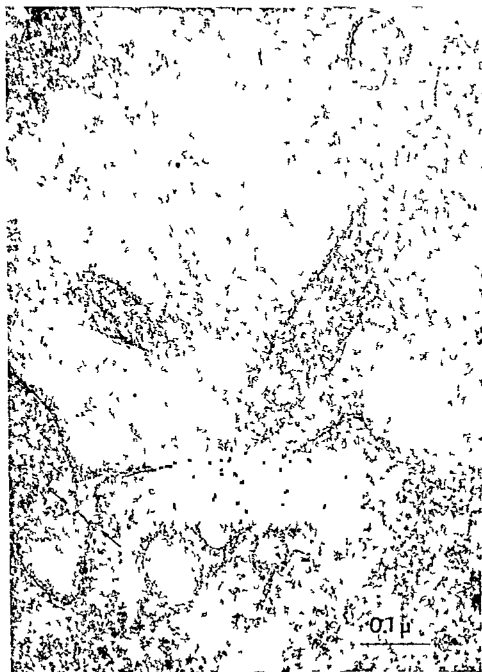



Fig. 50. The microvilli of the active light cells are long, up to 0.6 μ. They run in various directions and some have been divided transversely and others lengthwise. At their base invaginations are often found, sometimes almost closed off from the endolymphatic space with a fine membrane transversing their narrowest part (arrows). The fluid space appears finely granular in these regions. × 170 000.

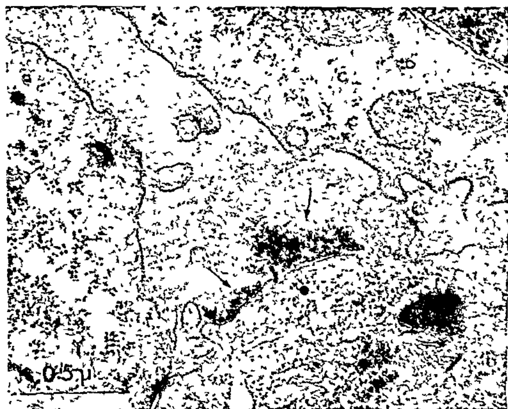



Fig 51 Silver particles can be seen in close contact with the surface of the dark epithelial cells (arrows) × 50,000




Fig 52 Protrusions from the apical border of the dark epithelial cells are often found encircling the colloidal particles The plasma membrane is often irregular with zones, where the three layered structure can not be clearly seen (arrows) × 95 000

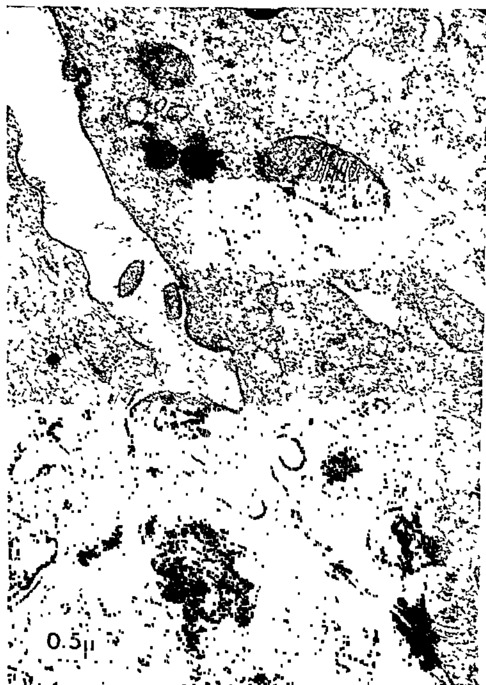



Fig 53 Finger like protrusions from neighbouring cells are sometimes found enclosing the colloidal particles × 41,000

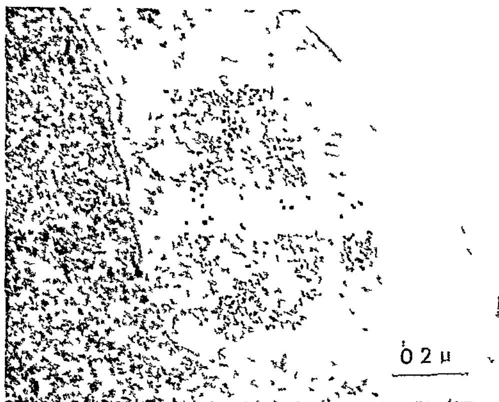



Fig 54 Silver particles are seen in unmembrane bound dilutions in the apical cytoplasm of the epithelial cells × 100 000




Fig 55 Inclusions containing silver could also be found in different parts in the cells and sometimes close to the basement membrane × 32 000

Fig 56 Red blood cells are seen being phagocytized by the dark epithelial ell 15 0 0

Fig 57 Cellular debris is often seen in close contact with the dark epithelial cells This debris can be seen partly invaginated into the cytoplasm and partly encircled by pseudopodia from the active cell × 60 000




Fig 58 Silver particles (arrows) can also be found in the connective tissue macrophages of the intermediate region of the sac × 15,000

## Subepithelial tissue

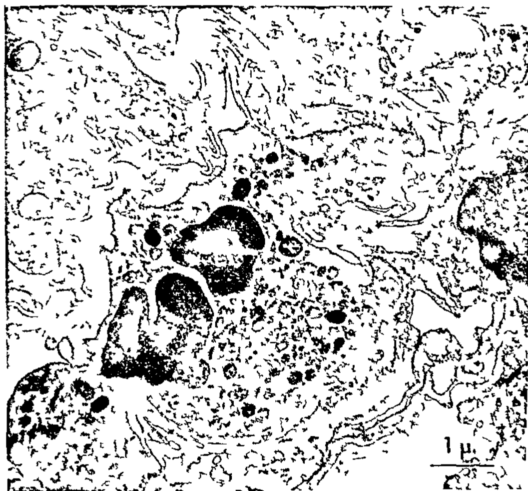
Changes in the connective tissue below the active region of the sac were seen

A slight increase in the number of leucocytes and connective tissue macrophages was apparent In some of these macrophages it was possible to see vacuoles as well as more dense inclusion bodies both containing silver granules (Fig 58 and 59) These inclusions were bounded by a unit membrane, as in the epithelial cells

The leucocytes were mostly free, very close to the epithelial lining and exhibiting signs of migration with pseudopodium-like protrusions (Fig 60) and could also be seen penetrating in between the epithelial cells (Fig 61)




F g 59 Deta l f om f g 58 show ng he nclu ons where s l er granules are found These n clus ons a e bounded by an un t membrane In he upper cell lamellar degenera on pa erns can be found (arrows) Collagen f brils a e ble between the cel s × 46 000




F g 60 Wander ng leucocyte close to the basement membrane of the ep thel um in the inter med ate port on of the sac × 15 000




ig 61. Wandering macrophages of different types could sometimes be seen to penetrate between the epithelial cells In this case a neutrophil leucocyte is demonstrated × 24 000

# Additional Experiments with Colloidal Silver Particles

A brief account will now be given of a small series of experiments performed to discover if colloidal silver particles could be found in the endolymphatic sac when injected by other routes, as has been suggested by earlier investigators, who injected dye intraperitoneally, intravenously, etc These investigators found an accumulation of the dye in the endolymphatic sac and perisaccular tissue (Andersen 1948, Engstrom and Hjorth, 1950, van Egmond and Brinkman, 1956)

## *Intravenous Injection*

An incision was made over the right external jugular vein of two of the animals and 0 5 ml of the colloidal solution was injected The animals were killed one hour afterwards

**Findings**

Silver granules were not found in the endolymphatic sac Some silver like precipitations were seen in the stria vascularis and in the vascularized region of the ampullary cristae, but the definite nature of these granules could not be established Degenerating nerve endings were seen in both the organ of Corti and the ampullary cristae, but the sensory cells appeared to be quite normal

## *Intracochlear Injection*

A hole was made in the scala tympani of the first cochlear turn in one animal and colloidal silver was injected A hole was also made in the scala vestibuli of the same turn in order to check that a free flow occured The inner ear was removed 18 hours after the injection In a second animal an injection was made into the cochlear duct via the basilar membrane but at the same time silver was inadvertently forced into the scala vestibuli of the first cochlear turn In order to see if the silver would enter the endolymphatic sac when Reissner's membrane was punctured, this animal was kept for 43 hours before the inner ear was removed

**Findings**

In both animals the organ of Corti was degenerated at the site of injection No silver granules were found in the endolymphatic sac of these two animals and no deviation from the normal situation could be observed in either the epithelial lining of the sac or its contents of free floating cells

# Injection of Bacteria into the Cochlear Duct

Living bacteria were injected into the cochlear duct of seven animals via the anterior approach into the basal coil of the cochleae A group C hemolytic streptococcus was used in six of the animals and the inner ears were removed at the intervals 10, 20, 20, 24, 24 and 40 hours after the injection E coli were used in one animal and the ear was removed after 24 hours

Five animals were examined with the electron microscope and two with the light microscope using serial sections according to the celloidin technique

## *General Findings*

The guinea pigs injected with bacteria suffered a much more vigorous general reaction than those injected with silver After about 20 hours they showed signs of labyrinthine involvement, such as moving very slowly around in their cages, heads tilted towards the operated side and disappearance of the Preyer reflex

*Middle ear*

The thick opaque yellowish tinted pus consisting of inflammatory exudate was seen filling the round and oval window areas completely concealing the stapes, in the operated ear of the animals injected with streptococci (Fig 62)

*Cochlea*

The cochlear duct was packed with inflammatory cells and bacteria In the lower turns of the cochleas the organ of Corti had disintegrated but in the apical coil it was possible to find areas much less affected Here the rows of haircells were intact and only a few inflammatory cells were seen near the tectorial membrane and stria vascularis The perilymphatic space, especially the scala tympani was almost completely filled with red blood cells and fibrous matter (Fig 62)

*Vestibular apparatus*

The sensory cells appeared normal in the utricle and ampullae Inflammatory exudates were not found in the endolymphatic space of this region although an increased number of red blood cells were present

## *Findings in the Endolymphatic Sac*

Of the five animals examined with the electron microscope bacteria were found in the endolymphatic sac of two of the 'streptococcal' guinea pigs (killed 20 and 40 hours after injection) and in the coli injected animal In the other two animals (killed 10 and 20 hours after injection) bacteria were not seen in the sac

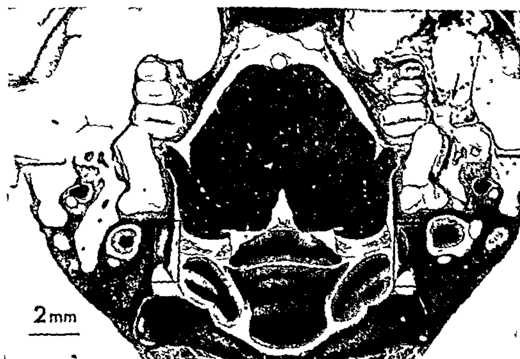


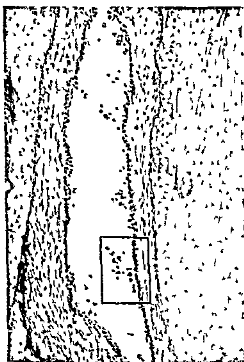

Fig 62 Low power view of both the operated and the control sides of an animal injected with *streptococci* The *middle ear on the right operated side is filled* with pus as is the cochlea (arrow) The endolymphatic sac on the operated side contains cells (right frame) whereas the control side has an empty appearance (left frame) For details see fig 63 *Heidenhain Susa* × 6

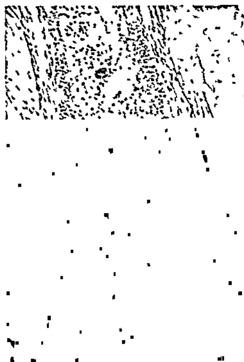
It thus seems that the most pronounced activity of the intermediate portion of the endolymphatic sac was found after approximately 20 hours analogous to the silver experiments (Fig 63) The bacteria were however more difficult to identify than the silver particles because in degenerative stages they could not be distinguished from other cellular debris and inclusion bodies of a similar size, that is about 0 5 *u*

The degenerating bacteria were most easily recognized when their characteristic cell wall with the thin amorphous capsule like outer zone was found intact, together with the lightly stained nuclear zone and the septae indicating the dividing zone

## Free floating cells

The lumen of the sac was filled with free floating macrophages and blood cells mainly neutrophil leucocytes in which bacteria could be found in various stages of degeneration







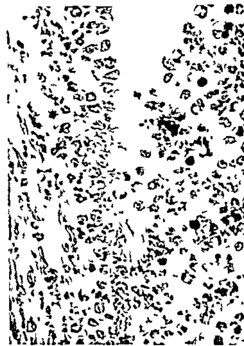



Fig 63 Detail from fig 62 The endolymphatic sac of the non operated side has an empty appearance with only a few rounded macrophages in the lumen (A) The epithelial lining is smooth and the connective tissue free from wandering leucocytes C The sac on the operated side is filled with cells of various kinds (B) consisting mainly of rounded free floating macrophages together with leucocytes The epithelial lining is irregular and the underlying connective tissue infiltrated with wandering leucocytes and other macrophages (D) *Heidenhain Susa* A and B × 110 C and D × 450




Fig 64 Degenerating bacteria could often be demonstrated in the rounded free floating macrophages The bacteria when seen in the membrane bounded vacuoles are undergoing degeneration (arrows) × 15,000




Fig 65 Detail of fig 64 The bacteria appear slightly more condensed than normal with little difference between the nuclear and cytoplasmic parts (1) In a slightly more degenerated stage light spots appear in the cytoplasm and their cytoplasmic border become irregular (2) In a still more advanced form of degeneration the outer capsule like part of the cell wall surrounds a thin and irregular bacterial cytoplasm (3) × 42 000

The bacteria were often found two by two in unit membrane bounded vacuolar inclusion bodies (Fig 64) In the first stages of degeneration the bacteria became more condensed with a nuclear region almost distinguishable from the bacterial cytoplasm The bacterial wall was seen as a dense zone just outside the plasma membrane followed by a thin amorphous and slightly less dense capsule-like zone at the periphery with a thickness of around 250 Å (Fig 65 and 66)

Later the bacteria had an irregular outline and cytoplasm varying in density which contained empty spaces Sometimes the bacteria appeared as being just on the verge of division with deep invaginations of their wall and a common zone of cytoplasmic and nuclear material between the symmetrical halves (Fig 65)

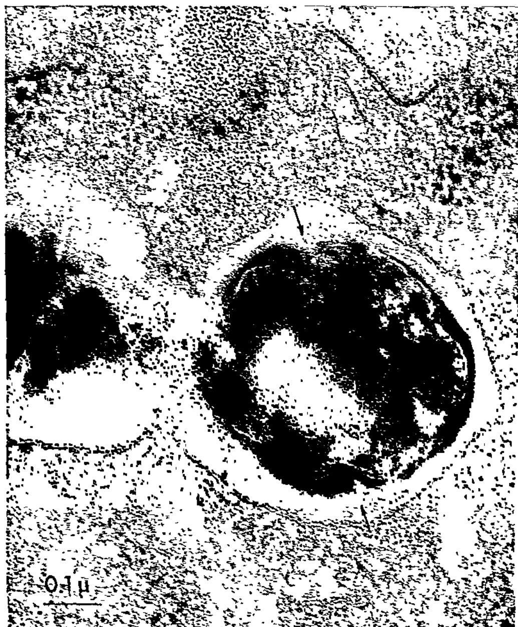



Fig 66. Detail from fig 64 showing an almost normal bacterium within a macrophage The bacterial cytoplasm appears very dense and it is difficult to distinguish the central nuclear zone. Outside the plasma membrane of the bacteria, a cell wall, consisting of a dark zone close to the plasma membrane and an outer zone of amorphous appearance, can be seen. Both zones are about 250 Å thick. Invaginations (septae) in the bacterial wall can be recognized at the periphery (arrows). The inclusion in which the bacteria is found is bounded by a triple layered membrane × 138,000.

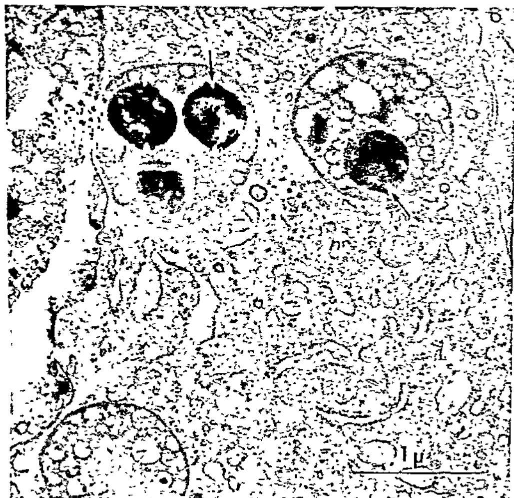



Fig 67. The bacteria are often found in large inclusions, up to 1 μ, the latter having a dense matrix and containing many vacuoles The macrophages with this type of inclusion has often a light cytoplasm. Note the clearly visible septae (arrows) × 32,000

# Discussion

Experiments attempting to elucidate the function of the endolymphatic sac have been performed by several investigators with conflicting results

In 1926, McNally destroyed the endolymphatic sac in rabbits, but did not find any changes in the subsequent labyrinthine function This type of experiment has been repeated by several authors, using monkeys and cats, and all agreed with McNally's original findings (Lindsay, 1947, Lindsay *et al* 1952, Schuknecht and Kimura, 1953) However, if the animal survives one year or longer, degenerative changes are seen at the extreme basal end of the cochlea in the organ of Corti (Schuknecht and Seifi, 1963) These could not have been the result of acoustical trauma from the burr, as damage from such a cause would occur much higher up in the cochlea These degenerative changes in the organ of Corti could indicate that destruction of the endolymphatic sac can impair the function of the inner ear without distention of the membranous labyrinth

According to the same authors, obstruction of the endolymphatic duct does not give rise to macroscopical or microscopical changes in the sac This appears to contradict the theories of earlier investigators (Boettcher, 1869, Hasse, 1881, Seymor, 1954, 1960) based on anatomical findings, that the endolymphatic sac secretes endolymph Schuknecht and Seifi were also unsuccessful in their attempts to irritate the endolymphatic sac to hypersecrete, by putting aluminium cream on top of the sac

As was earlier discussed, Guild (1927) has suggested that the endolymph was resorbed in the endolymphatic sac He injected potassium ferrocyanide into the cochlear duct and after tracing this substance to the endolymphatic sac, postulated a movement of endolymph from the cochlea towards the sac, where it was reabsorbed Several other similar investigations have been performed, such as that of Yamakawa (1929) who injected arsenious acid in the middle ear of rabbits and later showed its presence in the cells of the endolymphatic sac

Intravenous, intraperitoneal and subcutaneous injections of trypan blue by Andersen (1948) and Engstrom and Hjorth (who also performed intracochlear injections 1950), showed that the dye could subsequently be recognized in the lumen of the endolymphatic sac and the perisaccular tissue These authors believe that this was the result of small amounts of dye passing, via the blood stream, into the endolymphatic system It then circulated in the endolymph to the endolymphatic sac where concentration due to resorption occured They suggest that the intrasaccular cells then transport the dye particles through the saccular wall Other investigators however, believe that the dye is carried by the blood to the histiocytes in the connective tissue of the sac and from there into its lumen (Seymor, 1954 van Egmond and Brinkman, 1956)

The uptake of colloidal particles as demonstrated by electron microscopy has been studied in many cells and organs, such as the liver (Hampton, 1958, Hubner, 1962) blood capillaries (Wissig, 1958, Palade, 1960) subcutaneous macrophages (Muir and Golberg, 1961) cornea (Kaye, 1962 *et al*, 1962) pericardium (Staubesand, 1963) and in the synovial membranes (Ball *et al*, 1964) It has also been seen to occur in the ameba (Brandt and Pappas, 1962) In most of these investigations colloidal particles of heavy metals have been used

## *Experiment with Colloidal Silver*

The epithelial cells of the intermediate portion of the endolymphatic sac demonstrated an increased activity as compared with the normal endolymphatic sac, although the general features remained unaltered

### *Light cells*

The light cells showed an increase in the number of apical vesicles and microvilli, suggesting an increase in the uptake of fluid from the lumen of the sac In the base of these cells invaginations showed an increase in frequency although their general appearance was unchanged As discussed earlier, similar morphological findings have been described by several authors after studying cells active in fluid transport (Holmberg, 1957, Pappas *et al*, 1959, and others)

The light cells sometimes took up silver particles in a way quite similar to their normal pinocytotic activity, with silver granules close to the microvilli and in the small apical vesicles This way of taking up particles, by pinocytosis, has been described as being a feature of subcutaneous macrophages (Muir and Golberg, 1961)

### *Dark cells*

The dark cells exhibited pseudopodia which often embraced clusters of silver granules and cellular debris, to form membrane bounded vacuoles similar to those described by Gieseking (1958) in a study of the uptake of iron by lung alveolar surface cells Ball *et al* (1964) have described a similar phenomenon in the synovial tissue

The dark cells were on several occasions found in close contact with red blood cells, degenerating macrophages or cellular debris of unknown origin, demonstrating active macrophage activity Normal looking dark cells were also found occasionally protruding out into the lumen of the sac as if they were trying to leave the epithelial lining This might suggest that some of the free floating cells have been derived from the dark cells of the epithelial lining (cf Guild 1927, Kibata, 1927, Yamakawa 1929 Sirala 1941 Saxen, 1951)

It is interesting to note that a fibrillar cytoplasm has been described in the ameba *Chaos chaos* and has been related to cytoplasmic movements (Nachmias, 1964) These fibrils are very similar to those seen in the cytoplasm of the dark cells (Fig 15) They are not arranged in bundles or connected to the junctional complexes, as

explained by the slow movement of endolymph from the apical part of the cochlear duct towards the basal part and finally to the endolymphatic sac thus exposing the basal part of the cochlear duct to the ototoxic agents for a longer time

The reaction of the endolymphatic sac to the bacteria in these animals was very characteristic with an accumulation of cells in the lumen of the sac and large amounts of migrating leucocytes and histiocytes in the subepithelial areolar tissue moving towards the epithelium and through the widened intercellular space into the lumen of the sac This intense reaction from the subepithelial tissue was not encountered in the experiments with colloidal particles and must be regarded as the response of a reactive tissue to an inflammatory stimulus

This activity was most pronounced about 20 hours after the injection of bacteria which agrees with the findings of Ball *et al* (1964) where an inflammatory phase was demonstrated 18—48 hours after the administration of iron dextran into a joint cavity in rabbits

Some of the bacteria were found in the epithelium of the endolymphatic sac although it was more common to find bacteria inside the macrophages in the lumen of the sac. An explanation of this might be the very strict criteria used for identification of the bacteria Thus those bacteria which were engulfed by the epithelial cells might have been destroyed so much that it was not possible to adequately identify those inclusion bodies found

Macrophages containing bacteria were sometimes found in close contact with the dark epithelial cells in a similar way as when the red blood cells and silver containing cellular residues were phagocytized by the dark cells in the silver experiments

The bacterial degeneration could be closely followed in the free floating macrophages as a disintegration of the cytoplasm and nucleoid whilst the remnants of the bacterial cell wall could still be recognized inside the digestion vacuoles of the phagocytotic cells It seemed as if these bacteria often were slightly condensed and smaller than free bacteria These findings agree with those of Goodman and Moore (1956) and Goodman *et al* (1956) who studied phagocytosis of staphylococci by human leucocytes

It was not possible to trace the bacteria into the connective tissue macrophages as it was with silver as in their most degenerated forms they could not be distinguished from other cellular debris inside the various inclusion bodies

From the findings described and discussed in this study it seems that organic material such as bacteria and cellular debris is completely digested by the combined action of free floating cells and cells of the epithelial lining of the sac and that only the undigestible parts pass to the underlying macrophages

The studies of the normal structure of the endolymphatic sac and of the reaction to experimentally induced stimuli such as the effect of silver particles and bacteria suggest that the endolymphatic sac serves as an important reabsorbtive and reactive area in the labyrinth

The intermediate port of the sac with both light and dark cells in the epithelium seems ideally s t d for these functions

The *light cells* have a luminal surface studded with microvilli and pinocytotic vesicles and a basal part where invaginations are many indicating an increased surface area Thus this type of cell seems to be very well suited for the reabsorption of fluid

The *dark cells* contain various kinds of inclusion bodies and by using colloidal silver particles a marked macrophage activity was demonstrated by this type of cell

The areolar connective tissue deep to this epithelial lining was shown to react to a bacterial stimulus with an outpouring of histiocytes and wandering leucocytes which often squeezed themselves between the epithelial cells and into the lumen

The capillaries in this region also seem to be ideally suited for fluid passage as they contain many small pores in their endothelial lining

# Summary and Conclusion

The present work consists of two parts In the first the normal anatomy and ultrastructure of the endolymphatic duct and sac was described together with a brief description of the ultrastructure of the utriculo saccular ducts and the sigmoid sinus

In the second part, a series of experiments were described in which injections of colloidal silver particles and bacteria were made into the endolymphatic system and the reaction of the endolymphatic sac to this material was studied

## *Normal Ultrastructure of the Endolymphatic Duct and Sac*

**Epithelial lining**

***The endolymphatic duct*** has an epithelial lining consisting of squamous or low cuboidal cells

In *the proximal portion* of the endolymphatic sac the epithelial cells increase in size and constitute the transition between the duct and the following active intermediate portion of the sac

These cells do not show any marked signs of specialized activity

*The intermediate portion*

The epithelial lining consists of rather tall cylindrical cells irregularly arranged in protruding papillae or crypts

Two types of cells can be recognized located at random

*The light cell* is regular with a rounded nucleus of medium density, containing a nucleolus The cytoplasm has little density and is richly provided with endoplasmic reticulum, ribosomes and oval shaped mitochondria Many pinocytotic vesicles and vacuoles are present in the apical part of the cell, together with many long microvilli and various inclusion bodies The basal border of the cell is smooth but contains a few areas with characteristic infoldings surrounded by small vesicles which give an increased basal surface The side walls contain a tight junction, an intermediate junction, and one or more desmosomes The cell membranes interdigitate freely below these junctional complexes

*The dark cell* is often irregular with an elongated dense nucleus with invaginations at the nuclear border which almost fill the entire cell The cytoplasm is dense, fibrillar in appearance and contain the same organelles as the light cell although reduced in number The apical cytoplasm is not very richly provided with pinocytotic vesicles and microvilli but sometimes it contains large dense inclusions The basal border is often narrow and wrinkled with cytoplasmic papillae extending into the subepithelial tissue

*The distal portion*

The epithelial lining gradually changes from a cylindrical into a cuboidal type Both light and dark cells can still be found but here the light cells predominate In the extreme end of the sac only denser cells are present with an appearance similar to those of the endolymphatic duct

## Subepithelial tissue

The connective tissue of the endolymphatic duct and sac is of an areolar type which in the intermediate portion of the sac contains a rich network of capillaries close to the epithelial lining These capillaries often exhibit pores in their endothelial lining suggesting a transport of fluid

## Normal contents of the lumen of the endolymphatic sac

In the intermediate portion of the sac the lumen is irregular in form and contains cellular debris together with many free floating macrophages which are rounded and exhibit a marked pinocytotic activity Different types can be recognized one with a light cytoplasm and many dense inclusions another with a dense cytoplasm filled with large almost confluenting vacuoles giving a signet ring like appearance Various blood cells mostly neutrophile leucocytes are also present

## Utricular and saccular ducts

These ducts connect the endolymphatic sac to the utricle and saccule

The cuboidal epithelial cells are smooth and have a rounded centrally located nucleus The cytoplasm is of medium density with an even distribution of ribosomes endoplasmic reticulum and mitochondria but inclusion bodies are not present The cellular borders are smooth

The subepithelial tissue is of a loose type containing a few fibroblasts and scattered pigment cells

## Sigmoid sinus

The endothelial lining consists of elongated thin cells having a nuclear region bulging into the lumen

The nucleus is extremely wrinkled and surrounded by endoplasmic reticulum Golgi apparatus mitochondria etc The apical cytoplasm is also wrinkled in this region and shows many small pinocytotic vesicles and microvilli Otherwise the cell borders are smooth

The subepithelial tissue contains a few smooth muscle cells close to the epithelial lining and further below it blends with the areolar tissue of the endolymphatic sac

## *Experiments on the Phagocytotic Activity of the Endolymphatic Sac*

The aim of this part of the investigation was to study the reactions of the endo lymphatic sac when foreign material and bacteria were introduced into the cochlear endolymph

### Material and methods

A solution of colloidal silver was injected into young guinea pigs whilst hemolytic streptococci were injected into others

The microinjections were made into the basal turn of the cochlear duct

### Injection of colloidal silver into the cochlear duct

Silver particles were injected into the cochlear ducts of nine guinea pigs and specimens were examined 5 to 48 hours afterwards

The animals did not exhibit any signs of labyrinthitis but behaved normally after the operation In seven of the animals silver granules were present in the endo lymphatic sac, most pronounced after approximately 20 hours

#### *The endolymphatic sac*

The lumen of the sac was filled with free floating macrophages many of which containing silver

*The light cells* exhibited increased pinocytotic activity with many microvilli pinocytotic vesicles and vacuoles The characteristic basal infoldings were more prominent than in normal specimens

*The dark cells* demonstrated a marked phagocytotic activity Cytoplasmic protrusions were seen reaching out and encircling both colloidal particles and cellular debris of various kinds and incorporating them into unit membrane bound cytoplasmic inclusions

*The subepithelial tissue* exhibited a slight increase in cellular contents and silver particles could be seen undergoing phagocytosis by connective tissue macrophages

### Additional experiments with silver particles

Silver particles were injected intravenously or into the perilymph to find out if they could be transported to the endolymphatic sac via the blood or perilymphatic space

Silver particles could not be demonstrated in the endolymphatic sac or in it connective tissue in any of the animals injected

### Injection of bacteria into the cochlear duct

Living bacteria were injected into the cochlear duct of seven guinea pigs and the specimens were examined after 10—40 hours

The animals were affected with severe labyrinthitis The middle ear and the cochlea on the operated side were filled with pus

As in the silver experiment, the height of activity was reached after approximately 20 hours

*The endolymphatic sac*

The lumen of the sac was filled with free floating macrophages and leucocytes many of them phagocytizing bacteria These bacteria were seen in various stages of their degeneration

*The epithelial cells* contained many inclusion bodies which occasionally contained bacteria, and showed an increased pinocytotic activity Leucocytes and macrophages from the connective tissue, were often found penetrating between the epithelial cells of the intermediate portion

*The subepithelial tissue* showed a marked increase in cellular concentration containing connective tissue macrophages and leucocytes of various kinds

Identification of injected bacteria

In one of the animals injected with streptococci, thin slices of the endolymphatic sac and the cochlear duct were smeared onto blood agar plates which were subsequently cultivated Cultures of bacteria on the plates from both the cochlear duct and the endolymphatic sac showed that the organisms were identical to those initially injected

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Experimental and morphological evidence indicate that the main function of the endolymphatic sac is to act as a reabsorptive and a defensive mechanism for the inner ear The light and dark epithelial cells of the intermediate portion of the sac respectively reabsorbing endolymph and digesting cellular debris and foreign material in the endolymph

The macrophage activity described is partly seen in the dark epithelial cells and partly in the free floating cells Foreign matter and cell debris are to a large extent taken up directly by the epithelial cells with comparative little leucocyte reaction To live bacteria, the endolymphatic sac responds with an intense inflammatory reaction demonstrating typical leucocyte invasion and less activity of the fixed epithelial macrophages

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OTO-LARYNGOLOGICA

S U P P L E M E N T U M 202

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*From the Department of Audiology and Speech Sciences, Purdue University, Lafayette, Indiana*

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 202

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# INTRODUCTION

The occurrence of tinnitus aurium in patients seen by otologists and audiologists is extremely common Fowler (1944) (15) estimated that eighty-five per cent of the patients seen otologically complained of this symptom Heller (1955) (24) has stated that tinnitus is often the presenting symptom At least one important part of the rehabilitation for these patients is the management of such symptoms

Basic to the effective management of tinnitus aurium is an understanding of the causes of it In the case of tinnitus a complete understanding of causation does not appear possible as yet A collation of the available information, however, can greatly increase our understanding of tinnitus Before entering into the discussion of this topic, it is necessary to agree on a set of terms concerning tinnitus that will allow us to separate various subcategories of the symptom

The term *tinnitus aurium* means literally ringing of the ears, and will be used to refer only to sounds originating in the ear It will not be restricted, however, to sounds described as "ringing" but will include all sounds originating in the ear Head noises also may originate in the cerebral cortex In this case they are designated by the term *tinnitus cerebri* (Goodhill, V, 1950) (18) For the remainder of this paper, the term *tinnitus,* when used alone will refer only to tinnitus aurium Tinnitus may be defined further as a sensation of sound for which there is no source of vibration outside the individual There are two basic types of tinnitus aurium One type can be heard, under certain conditions, by an observer The other type is perceived only by the patient The first has been commonly referred to as vibratory or objective tinnitus The latter type being called non-vibratory or subjective tinnitus This report will be limited to a discussion of the latter type—non-vibratory tinnitus

*Purpose* This paper will attempt to present a comprehensive survey of the material published in English dealing with non-vibratory tinnitus When this literature has dealt with medical procedures and techniques, there has been no attempt to evaluate them When disagreement has been encountered concerning a specific procedure or technique, the various professional opinions and the relevant evidence on both sides has been cited without evaluation

# I. HISTORICAL OVERVIEW

In examining the history of the symptom of tinnitus aurium, one has the feeling of also following the development of medical philosophy The explanation of and the therapy proposed for tinnitus at various periods do not differ in any major way from the attention given to other diseases From the founder of medical ethics, Hippocrates, we may trace the efforts to understand the subject Engstrom and Graf (1952) (12) state that

aural murmur was a popular theme among the doctors and therapeutists of older epochs Hippocrates (c 400 B C ) took into consideration the possibility of an autoauscultation of the blood stream while A Cornelius Celsus (at the time of the birth of Christ) associated aural murmur with colds, headaches, epilepsy or other serious illness without discussing the cause of the noise Galenus (130–201 A D ) has also treated sonnitus in auribus but in much more antiquated reflections

During the next 1500 years, we may surmise that the status of knowledge concerning tinnitus followed the general medical practice of the time During some periods these head noises were without doubt treated as the entry of the devil into the body of the patient, hence, treatments associated with driving out the devil were employed As recently as a century ago there was a mixture of the modern and the ancient in the practice of medicine, and thus in the treatment of tinnitus aurium One of the writers of medical texts of that period, John Nottingham (1857) (38) expressed relatively scientific opinions He felt that "true' tinnitus aurium was that which was ' met with in cases where diseased conditions tell *upon*, or extraneous matters interfere *with*, the membrane tympani, the Eustachian tube, the ossicula, or the chorda tympani nerve " At the same time he recognized the existence of what is now termed subjective tinnitus and cited the need for a better understanding of the tinnitus that was associated with the inner ear The interesting link with the past in this comprehensive discussion of the diseases of the ear was Nottingham's advocacy of the use of leeches in the treatment of almost every type of malady described' Many cases of recovery are enumerated but one wonders if these were cures or rather the patients' efforts to bring a stop to the treatment

By the end of the nineteenth century, the only real progress toward an understanding of tinnitus was the reporting in the various medical journals of cases demonstrating the symptom and the describing of methods used in its treatment During this period the focal point of the symptom, in the minds of those writing about it, was the middle ear Houghton (1897) (26)

believed that "inflammation of the middle ear is the greatest factor in the causation of tinnitus aurium—acute catarrhal inflammation" This gave rise to Campbell's (1897) (7) interesting explanation of the mechanism responsible for tinnitus aurium

In cases of tinnitus aurium, which resist ordinary methods of treatment, perforation of the drumhead may be of great service, for it has been generally observed that there is usually not much tinnitus when the drumhead is perforated This gives rise to the accepted explanation that the noises in the middle ear disease, with imperforate membrane, are due to the obstruction of the outward flow of the sound waves and consequent concentration and reverberation in the tympanic cavity.

Also at this time there is the appearance in the literature of the first attempt to measure the frequency and intensity of vibratory tinnitus Cowan (1897) (8) found that in one patient the sound was in a frequency range of from 500 to 700 cycles per second (cps) and was of variable loudness He felt that the tinnitus in this case might be caused by a "filament stretched across the lumen of some blood vessel, which was thrown into rapid vibration by *the blood current" The course of treatment which resulted from* this theory was the continuous application of pressure to the carotid vessels The discomfort that this treatment caused the patient proved to be too great, however, and it had to be discontinued Another interesting case was reported by Green (1890) (21) at this time which showed one possible cause and successful treatment for objective tinnitus The diagnosis was one of hypertrophic changes in the nasal cavity The treatment consisted of surgery to remove the growth, and this surgical treatment was successful in terminating the tinnitus completely The probable explanation of the causation in this case was the pinching of a blood vessel in the area around the nasal cavity

During the period around the turn of the twentieth century, most of the articles in the literature were in the form of reports of clinical cases There was little, if any, effort made to generalize from the specific in formulating theoretical concepts for the understanding of tinnitus A great many physicians advocated many methods of treatment which were all more or less successful All of the treatments and hypotheses concentrated on the outer and middle ear and made no mention of the possible role of the inner ear in the production of tinnitus This was due in part to a lack of knowledge in the early 1900's about the role of the inner ear in the hearing process The situation was such that Delavan (1910) (10) remarked that "no substantial advance has been made with regard to the treatment of tinnitus in the last twenty-five years" However frustrating this period might have been to such men, it marked the turning point in the science of medical and clinical research

The second decade of the twentieth century marked the emergence of modern scientific thought in the efforts to improve understanding of the

symptom of tinnitus aurium For the first time tenable theories are found which explain the mechanism by which middle ear involvement could cause tinnitus One of these developed by Lothrop (1923) (35) concerns the action of the tensor tympani and the stapedius muscles in abnormal displacement of the ossicular chain This theory still has implications for understanding the total process of tinnitus and will therefore be discussed in detail in the *chapter on the etiology of non vibratory tinnitus From this start* towards a workable and scientifically sound explanation of tinnitus aurium more and more authors have offered their thoughts on the subject Unfortunately, there are some who have titled articles in a manner indicating that they intend to discuss their findings on tinnitus only to sidestep the issue be making no more than a brief mention of the condition For example, Hayes (1939) (23) wrote, 'I shall take up the question of tinnitus very briefly Tinnitus is a very baffling symptom'

A study of the history of thought concerning tinnitus is rewarding for two reasons First, it gives one an insight into the development of medical science through the years Second and more important in the scope of this work, the indication that tinnitus aurium is a symptom picture and not a disease entity becomes apparent That this is the case has been stated time and again in the literature *The variety of treatments that have resulted* in cures is legion The treatments involve almost all parts of the anatomy For example Campbell (1897) (7) writes 'I know of two cases of troublesome noises in the head which were cured by rectal operations" Frequently, however, this bit of knowledge—that tinnitus is a symptom—has been laid aside *when investigators started to look for the causes of tinnitus If* it were a disease entity one would approach its study by looking for one set of circumstances appearing in the histories of all who suffer from it In investigating tinnitus, such an approach has always met with failure When considering tinnitus as a symptom however, it is necessary only to find a common mechanism by which the several pathologies underlying tinnitus act upon the neural pathways to cause the perception of sound by the individual If this criterion can be met and the mechanism of tinnitus understood the course of effective and reliable treatment can be formulated

# II. ETIOLOGY OF NON-VIBRATORY TINNITUS

*General Considerations* The completely individualistic nature of non-vibratory tinnitus makes its evaluation most difficult The characteristic of individuality hinders the effective progression of one's study of this type of tinnitus whether it be in terms of an individual who wants treatment or in attempting to reach generalized conclusions about the problem The symptom is not one that an investigator may observe directly The patient may describe his tinnitus in terms of what it sounds like to him The terms used, however, may not call up the same images for the researcher that they elicit for the patient The patient may find that his tinnitus varies from day to day in pitch and loudness With an audiometer the patient may use a loudness balance and frequency matching technique to locate pitch and loudness of his tinnitus Since an individual's perception of pitch and loudness for the tones produced audiometrically vary daily, two variables are present in measuring tinnitus by loudness balancing

*Although study has been made more difficult by the subjective nature* of non-vibratory tinnitus, it has by no means been rendered impossible There are some things that may be postulated in commencing the study of subjective tinnitus First is the law of the specificity of sensory nerve fibers, which in the case of hearing means that when the auditory nerve is stimulated the only elicited sensation result can be hearing and that hearing will not result unless those areas of the cortex that contain terminal radiations of portions of the auditory nerve are stimulated Secondly, this type of tinnitus is not observable by anyone other than the patient Third, it can be stated on the basis of clinical observation that this type of tinnitus is more disorganizing to the patient than vibratory tinnitus This is true even though the level as measured by loudness balance techniques is often only a few dB above threshold (Sataloff, J J, 1949) (44)

Finally we may note that several authorities agree that there is a connection between deafness and tinnitus Kopetzsky (1948) (31) says that tinnitus always accompanies deafness in adults Saltzman and Ersner (1949) (43) believe that tinnitus is constantly present in the active phase of otosclerosis These are rather extreme positions and ones that most writers do not take Fowler (1944) (15) states that 85 per cent of 2000 consecutive patients with hearing losses reported having tinnitus Heller (1955) (24) has found that tinnitus appears in about 75 per cent of all cases of deafness he treats Table 1 is modified after a table by Heller and shows the per

TABLE 1. *Percentage frequency of tinnitus by type deafness.*

| Type | Present | Absent |
|---|---|---|
| Perceptive | 70.9 | 29.1 |
| Conductive | 65.0 | 35.0 |
| Combined | 87.5 | 12.5 |
| Otosclerosis | 85.0 | 15.0 |

cent of incidence of tinnitus as found in four diagnostic categories of deafness The figures show a consistently high incidence in each group The slightly lower figure in conductive cases may be due to the inclusion of some children in the statistics The significance of such an inclusion will be discussed later in connection with the relationship of tinnitus to age The statement is often made that tinnitus never occurs independently of deafness If deafness is thought of as a reduction in the ability to hear, deafness always accompanies tinnitus The masking effect of the tinnitus at the frequency or the band of its occurrence will always result in some reduction in the hearing sensitivity Usually, however, the tinnitus will account for only a small portion of the total hearing loss

The following four facts concerning non-vibratory tinnitus provide a starting point in our study of this form of tinnitus First, subjective tinnitus is to be found only in cases of hearing loss, although it is possible that the loss may be caused or increased by the masking effect of the tinnitus Second, the investigator may not observe the symptom directly Third, the tinnitus need only be of slight intensity to be annoying to the patient Finally, only the auditory neural pathways can transmit an impulse that will be perceived at the cortex as sound Though all four points are true, only the final one gives us any clue as to where to begin our attempts to understand non-vibratory tinnitus

As each theory of etiology has been considered it has had to meet one criterion This requirement has been that the theory must provide in some way for the stimulation of the normal acoustic pathway at some point from the organ of Corti to the cortex It is felt that failure to satisfy this criterion is in itself failure to constitute an acceptable hypothesis

*Etiology of Non-Vibratory Tinnitus General Organic Theories*—Writers have postulated several distinct locations of lesions which might produce the symptom of tinnitus Heller and Bergman (1953) (25) state that an irritation of the auditory neural elements anywhere between the tympanic promontory and the cortex will result in tinnitus Goodhill (1950) (1[illegible]) agreed and gave an extensive list of the possible sites of abnormal stimulation together with a list of the possible types of lesions that may produce tinnitus Table 2 presents a modification of Goodhill's list It is well to point out that even this is not necessarily a completely comprehensive list The

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to the other Saltzman and Ersner (1949) (43) state this phenomenon of lateralization is cortical in nature Without making any statements about other types of clinical groups they feel that lateralization is the mechanism by which tinnitus is localized in otosclerosis They state that a trigger sound starts the tinnitus They postulate that it might well be that the trigger stimulus is a vibration at the lowest frequency of the hearing range and that the tinnitus is a harmonic of that tone An explanation for the continuation of the tinnitus over a period of time is not made by these authors This perseverance may be explained in the theory of Stevens and Davis (1938) (48) concerning the etiology of tinnitus They stated that

> the most satisfactory explanation of most cases of the persistent ringing in the ears called *tinnitus* is that certain hair cells or the nerve fibers connected with them become hyperirritable and discharge nerve impulses more or less continuously as a result of some pathological process This abnormal condition may be acute and temporary as the result of excessive stimulation by a loud sound or it may be chronic

This type of situation may be occurring in the fixation of the ossicular chain in otosclerosis as the result of an irritation at the oval window

*Action of Intratympanic Muscles*—It is postulated by some that a type of muscle vibration may at times produce subjective tinnitus

Normally all of the middle ear elements interact freely and the tensor tympani and stapedius muscles maintain a state of equilibrium between the various middle ear structures This static equilibrium is constantly being disrupted in one s everyday life The parts tend to return to a state of rest as any given stimulus is removed Lothrop (1923) (35) hypothesized that if

> pathological conditions supervene and cause a displacement of the tympanic membrane ossicles and ligaments or any adhesions form involving the sound conducting apparatus an abnormal position of the tensor tympani and stapedius muscles must result and the normal balance is destroyed

The two muscles continuously try to modify any displacement through contraction Their contraction is the result of innervation by a branch of the mandibular division of the trigeminal nerve in the case of the tensor tympani and a branch of the facial nerve for the stapedius It is these involuntary contractions that Lothrop (1923) (35) believes are responsible for the tinnitus

> Every physician has placed his stethoscope on a muscle while it is being voluntarily contracted and heard the intermittent vibration So also in one or both of these muscles of the ear there are vibratory impulses and thus this persistent intermittent vibration of the muscles acting against an unbalanced ossicular chain and elastic adhesions produces a vibration in the labyrinthine fluid

The vibration in the labyrinthine fluid produces the same effect as that produced by any other pressure pattern Thus the organ of Corti is stimulated producing a sensation of hearing which is tinnitus If the equilibrium

of the middle ear is re-established, the muscles will relax and the tinnitus will cease. This may result from a return of the middle ear structures to their original position. Certainly one cannot account for all cases of subjective tinnitus in this manner, since the frequency of the tinnitus is often of an order greater than the number of vibrations of which the muscles are capable. Lothrop does not maintain that all tinnitus is the result of the contractions of the tensor tympani and the stapedius.

Lothrop's theory is applicable even when, as in fenestration, the ossicular chain is removed. With these structures removed the vibration may still be produced through the action of the stapedius on the ankylosed footplate of the stapes in the oval window. Jones and Knudsen (1928) (28) share Lothrop's viewpoint that the structures of the middle ear may produce tinnitus.

This theory of muscular activity may well explain some of the observations of Saltzman and Ersner in their work with otosclerosis. The condition they describe is the same lack of equilibrium among the middle ear structures. The adhesions of otosclerosis result in an abnormal position or restriction *of the movement of both the tensor tympani and the stapedius muscles.* The fact that the tinnitus tends to disappear when the air conduction loss reaches a level in excess of 60 dB is explainable also. At this point the fixation of the ossicular chain and the ossification at the oval window is so complete that a new position of equilibrium is established and the contractions of the muscles cease.

It should be emphasized that all authorities do not agree with this explanation of the role of the tympanic muscles. For example, Atkinson (1946) (3) feels that "increased tension in the intrinsic muscles of the ear is not a factor in the production of tinnitus except in cases where an emotional disturbance is also present."

*Tympanic Plexus.*—On the promontory of the middle ear several nerves meet in what is called the tympanic plexus. This plexus is considered by Schneider (1947) (45) and Trowbridge (1949) (50) to be the source of non-vibratory tinnitus. Trowbridge describes the composition of the plexus and its role in producing non-vibratory tinnitus in the following manner:

> Through this connection of the trigeminus, sympathetic and glossopharyngeal nerves, which form the tympanic plexus, tinnitus aurium may be produced by pathological changes in the middle ear and in neighboring structures such as the teeth, pharynx, nose and eustachian tube ... the tympanic plexus acts as a central nerve plexus, receiving impulses through its connection with the ninth nerve, the fifth nerve and the carotid sympathetic fibers.

In Schneider's view, the tympanic plexus is the critical portion of an internal (vegetative) sound system. The theory is complex in nature and is part of a larger theory of the development of the entire nervous system. Only the portion of the theory that seems to be applicable to the present study will be presented here.

Of particular importance to an understanding of tinnitus is the portion of the theory that has to do with hearing Schneider maintains that there are two sources of sound—external and internal The external source is the one with which we normally associate hearing The internal system may also be important, however The possibility has been discussed earlier that one source of non-vibratory tinnitus may be the activity of the tympanic muscles This theory has relied on the normal conduction pathway of the inner ear and the organ of Corti for the perception of sound It is Schneider's (1947) (45) contention that "by way of the tympanic plexus sound *may be relayed to the, labyrinth, cochlea and to the brain stem, i e, to the* tractus solitarius, especially along the visceral afferent fibers of the seventh . and the ninth [nerves] " He believes that the internal system may in this manner bypass the usual pathway at the peripheral levels If this is true, it serves to explain some of the most puzzling problems in tinnitus First, if tinnitus is passed along the normal auditory pathway, section of the auditory branch of the VIIIth nerve should give relief It is estimated that such relief is obtained in only about 30 per cent of the operated cases The theory of a continuous stream of impulses flowing from a cut nerve was cited by Fowler (1939) (13) to account for some portion of these cases not receiving benefit from this surgery *It is hardly* conceivable, however, that all such cases result from this phenomenon The concept of a vegetative sound system also helps to explain how tinnitus may result from conditions that do not appear to be associated with the ear at all Among the conditions that Schneider (1947) (45) mentioned as possible causes of tinnitus are defective teeth, food allergy, and various nutritional deficiencies

The mechanism by which the process of hearing is maintained in equilibrium is explained by Schneider by separating hearing into two systems The external sound reception system (E) and the internal sound reception system (I) Each system is composed of three levels—the cortical level (c), the brain stem nuclear level (n), and the peripheral level (p) At a given time E(c+n+p) may be greater than, equal to, or less than I(c+n+p) The normal situation is for E to be greater than I (E>I) In the normal *state, the external and internal systems have connections at the brain stem* and at the peripheral levels These connections are shown diagramatically in Figure 1, adapted from Schneider The normal pathway (left side of figure) is a special sensory tract that progresses directly from the peripheral to the cortical The remainder of the diagram shows a double circuit between the internal and the external systems This is to be expected when one remembers that the internal system has other primary functions that involve more complex inter-relationships However, the internal system does normally follow a one way path when considered only as a sound transmitting system This is from the viscera through the tympanic plexus to the cephalic ganglia and terminating in the area of the labyrinthine and auditory nuclei Schneider states that the connection between the plexus and the organ of

Corti is probably not direct, but rather it is through the control of the blood supply to the organ of Corti that the relationship between that and the plexus is accomplished

If Figure 1 is a true picture of what happens in the normal situation, several illuminating assumptions may be made concerning the various pathologies of the ear. In the case of otosclerosis, the fixation of the stapes results in a reduction of the intensity of $E_p$ and therefore a reduction in $E_n$ and finally in $E_c$ This reduction of the external system, although it does not make the internal system greater than the external (at the cortical level) allows the internal to be close enough in intensity to be perceived As $E_t$ is reduced further and further there is a corresponding increase in the relative intensity of $I_c$ in relation to $E_c$ It has been noted that at somewhere around the 60 dB level of loss of air conduction the perception of tinnitus tends to cease Schneider believes that tinnitus disappears as a result of the degeneration of the tympanic plexus due to sclerosis of the promontory of the labyrinth

The relationship of these two systems is then the basis of the theory of Schneider He feels that the function of the internal system is to support the external system The external system carries the meaningful sounds of the environment, and it combines with the internal system to act as a single wave form at the cortical level

The question may be raised as to how Schneider would explain why tinnitus does not continue to increase until total deafness is reached and remain after the external sound system is completely inoperative The answer is that it is not the deafness itself that causes tinnitus to cease Rather it is the degeneration of both of these systems, both at the peripheral and labyrinthine nuclei levels, that results in the cessation of the tinnitus and in total deafness Schneider concludes that deafness and tinnitus represent a disease of the cranial parasympathetic system Support for Schneider's view of the importance of the plexus is found in Lempert's (1946) (32) statement that at times tinnitus "may be due to a tonus impulse transmitted to the inner ear by diseased sympathetic ganglion cells of the tympanic plexus"

The neurological evidence for such an interpretation is as yet inconclusive and acceptance of Schneider's viewpoint must await further proof of his assumptions

*Inner Ear Pathology* —Whenever hearing function is discussed, one normally thinks of the inner ear, and particularly the organ of Corti This organ is one of many small structures in the body which are vital to everyday life and about which little is known It is believed that the cilia of the organ of Corti are the first link in the neural portion of the auditory system The exact mechanism by which changes in pressure within the fluid of the inner ear cause nerve impulses to be initiated is not clear It may be through a chemical discharge, or a piezoelectric type of action The stimulati[illegible] these cilia in an abnormal manner may take place due to a

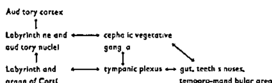



FIG 1 Normal condition

hyper-irritability of the type described by Stevens and Davis (1938) (48) Morrison (1938) (37) states that this irritation is the result of "degenerative vasomotor and vascular changes"

Fowler (1940) (14) suggests that the frequency of the tinnitus may coincide with the edge of the lesion on the organ of Corti This he explains by the principle of the greatest sensitivity occurring at the junction of two extremes of sensory perception This principle is demonstrated when one puts an arm into very hot water and is aware of heat most intensely at the level of the surface of the water Somewhat similar to Fowler's concept is that of Atkinson (1944) (2) He views tinnitus as an auditory paresthesia in which the nerve endings of the cochlea are being stimulated in the same manner as those in other parts of the body during the sensation of itching

It is extremely difficult to obtain definitive information on the function of the organ of Corti or on the workings of the cochlea itself In post mortem examination of the inner ear there is usually so much degeneration of the structures that it is not possible to determine the relationship of the deafness and tinnitus to the lesions found One bit of evidence seems to confirm the thinking which holds that the frequency of the tinnitus and the edge of the lesion coincide This is the statement by Davis and Fowler Jr (1960) (9) that on the audiogram the frequency of the tinnitus will often be found at the point where the loss increases rapidly "Here it probably represents a local irritation, the first stage of a degenerative process that has already destroyed some of the neighboring sensory cells" More recent information (Graham and Newby, 1962) has indicated that this relationship is not found as frequently as might theoretically be predicted

*Psychosomatic Theories* —The interdependence of the mind and the body in maintaining general health and well being has been studied extensively in recent years The possibility of a connection existing between the emotional state of the individual and the onset of tinnitus and deafness has also been examined It would appear that there are two ways in which the influence of the emotions on hearing could take place These are the effects of emotion both on the fluids of the inner ear and on the blood supply to the same area At present not enough is known about endolymph or perilymph to draw any conclusions about how they might be influenced One might theorize that emotional states cause the salinity of the endolymph to increase and thus create a condition of irritability in the organ of Corti

The material written thus far on this phase of tinnitus deals with the blood supply to the ear

Fowler and Fowler (1950) (16) have developed this theory to explain how "psychoneurovascular factors cause tinnitus . " They stated that emotional factors may cause the same somatic results as shock "After traumatic shock . and in all severe infections and toxic diseases, thick agglutinated masses have been observed to form The masses are called 'sludges' " In large vessels these masses do no harm and pass through the body to the liver and the spleen where they are broken down In organs such as the cochlea, the blood vessels are extremely small and the sludges are unable to pass through Thus there results a reduction of the blood supply to the nerve endings This lack of blood works as an irritant to the nerve cells "It should not be surprising if irritation so caused resulted in tinnitus, and if prolonged eventually resulted in degeneration in the neural tissue " From a physiological standpoint this seems quite a reasonable theory For the theory to be cohesive, however, a cause and effect relationship would have to be demonstrated Fowler and Fowler (1950) (16) propose two steps for correlating the emotional factors and the appearance of the bodily symptoms

1 Establish a time relationship between the emotional episodes and the onset or exacerbation of symptoms (We are herein concerned with tinnitus and deafness in otherwise apparently healthy persons )

2 If no correlation is remembered or recognized, or no memory remains of the emotional upsets yet the patient is and has been hypersensitive to emotional stimuli then it is highly probable that emotion was a factor in the etiology There is *nothing else more probable*

Three factors seem to be important in determining whether or not blood sludging will produce tinnitus First, there are different types of sludge, some of which are more likely to produce tinnitus than others Second, the length of time that the sludge remains in the system is important Finally as in all pathology, the prior health and condition of the organism affects its susceptibility to disease

The sympathetic nervous system controls the size of the blood vessels Passe (1953) (40) reports that stimulation of the sympathetic nervous system tends to act as a vasoconstrictor in the case of the ear Thus when the body presents its defensive mechanism of fright the sympathetic nervous system causes a reduction in the blood supply to the ear This reduction may have the same effect as the blood sludge of Fowler and Fowler's theory Once again the individual differences in human structure and bodily function would account for some people who, although they are subject repeatedly to such stimulation, do not develop tinnitus or become deaf The fact that such a vascular control is exerted has been shown by Passe (1953 (40) through the use of both stellate ganglia anesthesia and by upper dorsal sympathetic ganglia block The results of this technique have

been good in the reduction of tinnitus. It is believed that an increase in the blood supply has been responsible for this success.

It is entirely possible that the processes described in these two theories may be occurring simultaneously. If this is true the combination of vasoconstriction and blood sludging would serve to aggravate the reduction in blood to the inner ear.

*Psychoanalytical Theory.*—It is not feasible or particularly desirable at this time to go into psychoanalytical theory to any degree. The concept of past experience determining the particular manifestation that will appear in neurosis is central to the present discussion. Also of importance is the theory of the development of the super ego as an internalization of the values of society.

The ear and the function of hearing in the daily life of an individual is of great importance. Through hearing we maintain contact with the living world. It is through this sense as no other that we relate to the changes that are constantly taking place in the world around us. The process of adjustment to this ever changing environment is frequently a source of conflict for the personality. It would seem consistent with the various theories of personality problems that failure to adjust may result in the refusal of the organism to recognize auditory stimuli on the conscious level (hysterical or conversion deafness). It is also quite possible to distort the functioning of the organism on the cortical level. Weinshel (1955) (52) believes "there are, therefore, a number of psychological mechanisms which can be responsible for the production of tinnitus ..." As the individual is developing the super ego it is through the ear that he receives the "voice of authority" which is presented to him by various adult figures. This voice speaks in terms of right and wrong, and holds the power to transmit approval or to withhold love. When the dictates of this authority are violated, the individual may experience feelings of guilt. The guilt may evidence itself by the voice of the parent speaking to the individual in a symbolic way. The ear may then "hear" these voices in the form of tinnitus and thus receive punishment for wrongs. It has been mentioned earlier that although tinnitus measures only a few dB above threshold by loudness balancing techniques it is often perceived by the individual to be very loud. This recruitment phenomenon can be explained in terms of the punishment concept as the individual's attempt to atone for his wrongs.

In psychoanalytical thinking all of the bodily openings have sexual significance. The sexual life of the individual is central in the development of the ego and the super ego. The basic sexual conflict is that of establishing oneself as a member of one's own sex. During the adolescent period of development there is normally considerable ambivalence in this area. The roles of both sexes are commonly experimented with. In the symbols of sex the ear has the unique position of being both a protuberance and an orifice. Thus both the male and the female organs are embodied in the one structure. The ear may therefore play an important part in feelings

of guilt about sex and also in feelings of insecurity about sexual status In this regard Weinshel suggests that tinnitus fills the orifice that the ear represents Tinnitus in this way becomes the symbol of the male genitals or the sex act or both He has found that although these super ego problems *are not found universally in cases of tinnitus, or tinnitus found universally* in super ego problems, there are a large number of cases in which both are present Weinshel indicates that the combination of tinnitus and super ego problems occurs more often than can be explained on the basis of chance alone

# III TREATMENT OF NON-VIBRATORY TINNITUS

*General Considerations* The treatment of any disease or pathology is based upon an understanding of the dynamics of that condition When there is a wide range of opinion as to the etiology of a condition one would not expect unanimity as to the course of treatment to be followed Precisely such a situation exists in regard to the condition known as non vibratory tinnitus aurium Heller (1955) (24) has compiled a list of the various types of treatment that have been used and although this may not be a complete list it serves to show the range of treatment Table 3 is from Heller s list

Commenting on all of the items in Heller s list obviously would not be prudent or worthwhile Many of them are listed only because at one time or another someone received relief from tinnitus while on such a course of treatment In some of these cases the recoveries were probably spontaneous and actually unrelated to the therapy Several of the methods listed have been used fairly extensively and have been evaluated It is these therapies then that will be the focus for this section As was stated earlier the medical rationale for these treatments will not be discussed other than to point out the general basis for their use With this exception the discussion will be confined to the results obtained by the various methods

*Medical Treatments* One notable bit of investigation has occurred in the area of medical treatment This has been in the use of vitamin A injections given intramuscularly Experiments on animals have shown that diets deficient in vitamin A cause a degeneration of the cochlea and that massive doses of vitamin A will arrest the condition Lobel (1949) (34) found that this vitamin was also effective in improving hearing sensitivity and alleviating tinnitus in human beings Anderson Zoller and Alexander (1950) (1) found a high degree of agreement with these results as did Bau and Savitt (1951) (6) All of these studies showed both an improvement in the hearing sensitivity and a reduction of the tinnitus in a majority of the cases reported However subsequent studies by Baron (1951) (5) and by Atkinson (1954) (4) have failed to substantiate these findings The last two investigators have shown independently that this form of medication is not a cure all for tinnitus if indeed it is effective at all The results reported by various investigators are reported in Table 4 It has been suggested that some of the cases would have shown improvement with any treatment responding simply to the knowledge that someone was trying to help them This is an important point to keep in mind when setting up an experimental evaluation of any new therapy and points to the need for utilizing both control and experimental groups

TABLE 3 *Treatment of tinnitus*

| | |
|---|---|
| *Medical* | |
| 1 | Medication: bromides, barbiturates, other sedatives, potassium iodide, vitamins, benzyl cinnamate, antiallergic drugs, histamine therapy, intravenous procaine |
| 2 | Local therapy to disease process |
| 3 | Elimination of drugs and intoxicants |
| 4 | Elimination of foci of infection |
| 5 | Correction of faulty gastrointestinal function |
| 6 | Correction of metabolic diseases |
| 7 | Control of diseases of the vascular system and blood forming organs |
| 8 | Dietary control of fluids, salt and water balance |
| 9 | Dental rehabilitation |
| 10 | Intratympanic medication |
| 11 | Therapy directed to correct nose and throat pathology, including roentgen and radium therapy |
| 12 | Politzerization, inflation, massage |
| 13 | Removal of cerumen |
| 14 | Psychotherapy |
| 15 | Hearing aid |
| 16 | Electric therapies, i.e., ultra violet, quartz lamps, galvanism |
| *Surgical* | |
| 1 | Otologic: ossiculectomy, mastoidectomy, chorda tympani resection, fenestration of the labyrinth, obliteration of the saccus endolymphaticus |
| 2 | Rhinologic |
| 3 | Spinal tap |
| 4 | Cranial surgery for tumor, vascular anomalies, section of eighth cranial nerve. Stellate ganglion block |
| 5 | Splanchnectomy and similar technics for alleviation of hypertension |

Various other drugs have been used with what on the whole have been rather discouraging results. Fowler and Fowler (1950) (16) do report some success in alleviating tinnitus in psychosomatic cases with intravenous administration of procaine. It has been observed in other types of pathology exhibiting blood sludging that this treatment breaks up the sludge. Fowler and Fowler's success seems to confirm an earlier use of the drug by Lewy (1937) (33) in which he found that procaine hydrochloride was effective in reducing tinnitus in a large majority of the cases he treated. The length of time the relief lasted was variable, however. The effectiveness was particularly high in the case of deafness of the inner ear with only two cases failing to show a reduction in the intensity of the tinnitus. It also was observed that vibratory tinnitus was not relieved by this method of treatment.

In conjunction with his feeling that the tympanic plexus is a key in the etiology of tinnitus, Trowbridge (1949) (50) has found that tympanic injection producing tympanosympathetic anesthesia is a simple and practical procedure for the relief of tinnitus aurium and secondary otalgia in

TABLE 4 *Response of Tinnitus to Vitamin A Therapy*

| | Investigator | Number of Cases | Improvement in Tinnitus |
|---|---|---|---|
| 1 | Lobel | about 300 | Considerable number' |
| 2 | Andersson Zoller and Alexander | 23 | 39% |
| 3 | Bau and Savitt | 9 | 66.6% |
| 4 | Baron | 17 | 23.5% |
| 5 | Atkinson | 10 | none |

properly selected cases Trowbridge found that the best results were obtained in those cases where the treatment was begun early in the history of the complaint There were few side effects and no harmful after effects from the drug

Shambaugh and Jennes (1942) (47) found no conclusive evidence for the use of thiamine hydrochloride in relief of nerve deafness and tinnitus aurium The use of prostigmine has proved to be effective in alleviating tinnitus in only about 10 per cent of the cases on which it was used as reported by Juul (1946) (30)

Some success in treating tinnitus has been obtained on a temporary basis by the use of nicotinic acid as a vasodilator to increase the blood flow to the labyrinth Wille and Flottorp (1954) (53) and Atkinson (1944) (2) have reported on this method of treatment The latter study showed better results in those patients whose hearing loss was of the conductive type

Halton Erullar and Rosenberg (1960) (22) reported on the use of galvanic current for the relief of tinnitus The authors varied the polarity of the current by placing the anode or cathode on the zygomatic arch of the affected side When the anode was so placed the intensity of the tinnitus decreased as the current increased When the cathode was used increases in current were accompanied by an increase in the intensity of the tinnitus

*Surgical Techniques* There are two general surgical approaches to the treatment of tinnitus One is designed to increase the blood supply to the region of the labyrinth and the other is designed to remove the nerve pathways that might transmit the tinnitus

On the basis of the relationship between the sympathetic system and vasodilation or vasoconstriction Passe (1951) (39) advocated that the stellate ganglia or the upper dorsal sympathetic ganglia be blocked Following this procedure eight patients reported partial relief five had complete relief and three found no relief from their tinnitus In a later work published after his death Passe's (1953) (40) results were not so good In this study only 50 percent of the cases showed improvement

Johnson (1954) (27) also has used sympathectomy successfully in treating tinnitus He used a stellate ganglia block and if this reduced the tinnitus he performed the dorsal sympathectomy This procedure insures a rather

high degree of success, since the two operations apparently accomplish the same end result one temporarily and the other permanently The poor results obtained by Atkinson (1944) (2) in using the stellate block indicate that this procedure alone does not offer much hope for successful treatment in most cases

The investigation of the theory that the tympanic plexus is a focal point in the production of tinnitus has led to the other major area of surgical procedure Schneider (1947) (45) suggests that "the tympanic plexus be stripped in those cases where the plexus may be irreversibly damaged ..." He thinks that in cases where the "internal sound system" is overbalancing the "external" at the peripheral level, this procedure should eliminate the tinnitus At present, ascertaining the existence of involvement at the peripheral level is not normally possible, so the operation is not usually feasible Lempert (1946) (32) also has theorized about the malfunction of the tympanic plexus as a cause for tinnitus He feels that a tonus impulse can be transmitted to the inner ear from the diseased plexus, and that stripping the plexus may eliminate the tinnitus This operation Lempert called tympanosympathectomy. It was reported as being successful in improving the tinnitus for ten out of fifteen patients Although Lempert is acting on empirical findings, and Schneider's recommendations are made on the basis of a theoretical construct, both writers advocate the same general surgical procedure

There has been one report by Elithorn (1953) (11) of the use of prefrontal leucotomy (lobotomy) in the relief of tinnitus The cases reported were all being treated for psychotic conditions, and the operation was performed to relieve those conditions In fourteen cases, tinnitus was reported to be the primary symptom of the psychosis, and in the remaining three cases it was only a focal point of the psychosis The results of the study showed that nine made substantial recovery, four showed some improvement, three were unchanged, and one became worse The cases of recovery did not necessarily involve large reductions in the intensity or duration of the tinnitus, *but rather made the tinnitus less bothersome*

The fenestration operation and the stapes mobilization frequently result in the alleviation of tinnitus It seems that in these cases it is the improvement of the hearing sensitivity that results in the reduction of the tinnitus It is also possible that the tympanic plexus or some other area is involved in the operation, and the reduction of tinnitus may not always be attributed to the improvement in hearing that results from the operation

*Psychotherapeutic Measures* Those types of therapy or treatment that are not surgical or medical (with the exception of sedation) in nature are included in this category Often these psychotherapeutic measures will be used to supplement a medical or surgical treatment

Goodhill (1957) (19) has developed a list of five measures for the treatment of tinnitus

*Palliative Measures* Simple reassurance as to the reality of the tinnitus accompanied by encouragement and good prognosis will go far in helping to alleviate the anxieties of the patient with decompensated tinnitus

*Acoustic Sedation* Acoustic sedation is very helpful in many cases of tinnitus especially in regard to the difficulty in sleep which is a great problem with many patients The use of a bedside or pillow radio or phonograph speaker *is very helpful in providing an artificial source of ambient noise to mask out* the subjective tinnitus

*Drug Sedation* Drug sedation is an important palliative not only for daytime use but especially for bedtime tinnitus irritability No one drug should be used for a long period

*Surface Psychotherapy* Such surface psychotherapy should include a thorough explanation as to the real nature of tinnitus with assurance that it is neither hallucination nor an illusion

*Major Psychotherapy* Deep psychotherapy in psychiatric hands is indicated in every case of organized symbolic verbal or musical tune tinnitus It is also indicated where surface psychotherapy has not solved the decompensated tinnitus problem

Through these measures the effort is not so much to cure the tinnitus as it is to make it bearable The patient s acceptance of the tinnitus is described by the terms compensated and uncompensated tinnitus It was Goodhill s (1950) (18) goal to convert uncompensated tinnitus into psychologically compensated tinnitus In accomplishing this goal the five measures reported above are used in combination rather than singly Any approach to the treatment of tinnitus from a psychological viewpoint will utilize similar measures to those advocated by Goodhill Fowler and Fowler (1950) (16) report that the patient s attitude towards his tinnitus was an important factor in the success of any treatment

The use of a hearing aid for the relief of tinnitus has been studied by Saltzman and Ersner (1947) (42) They feel that the benefits obtained from a hearing aid are analogous to those of the fenestration operation By amplification much outside sound is enabled to reach the cochlea crowding out and masking the patient s head noises Although no statistics are cited Saltzman and Ersner believe that if a hearing aid can be worn *successfully relief from tinnitus will be obtained For many patients relief* through amplification is unavailable as the characteristic of their hearing loss precludes the use of a hearing aid

# IV. RELATION OF TINNITUS TO AGE

A study of the literature on tinnitus aurium has resulted in the finding of only a few isolated references to childhood cases. The type of distribution shown in Figure 3 by Ventus (1953) (51) is probably typical of the occurrence of tinnitus by age. For the study from which Figure 3 is taken, Ventus selected an isolated area in the British Isles. The population of this area *represented on a small scale the general population of the country.* The area was self sufficient and had farming, industrial, and metropolitan regions which paralleled on a small scale the country as a whole. This sample was small, but it included all the inhabitants of the area. By virtue of using the total population, the problems of random sampling were avoided. The author feels that use of a finite population might sufficiently offset the disadvantages of a small number of cases to allow for some generalization about tinnitus in the unrestricted population.

There are several observations that may be made from Ventus' data. Some are supported by other evidence, others are entirely in the form of conjectures and at present have no known support other than logic.

The first thing of significance that one notices on this graph is the first age group. Why does Ventus not report cases younger than fifteen? Fowler and Fowler (1955) (17) have found that "children, before puberty, seldom mention tinnitus or monaural deafness and even binaural hearing loss may be taken in stride. Children seldom mention ... any moderately severe symptom unless it is associated with pain."

*Young children focus attention on themselves just as they are curious* concerning all aspects of their environment. This attention to bodily functions during early childhood is not internalized in terms of a self evaluation. During the period of late childhood interest in the self wanes and is replaced by interests in the new events in the environment such as the school. With the advent of puberty there is a reawakening in the child of the earlier interest in the self. However, this time the awareness is much more ego involved as the child is becoming aware of minor differences between himself and others. The child searches himself for differences, real or imagined, that might set him apart from the group. Tinnitus is a subjective sensation that seems to be highly influenced by the psychological outlook of the individual. When the individual is looking for differences in himself, it is even more probable that tinnitus would be noticed if it were present.

The place of hearing in the development of the super ego has already been mentioned. The renegotiation of controls of the adolescent period is the

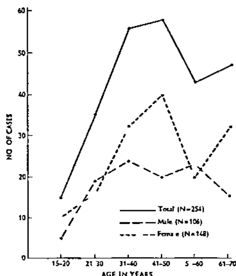



FIG 2 Incidence of tinnitus related to age

major source of guilt feeling for this age group These controls center about sexual and authoritarian topics Even if the previously stated hypothesis—that the individual hears the "voice of authority" in the form of tinnitus—is too strongly stated, it seems more than possible that there is some connection between the two One could draw several possible conclusions regarding this problem of balance between the external and internal sound systems from the formulas presented by Schneider (1947) (45) The most obvious would be the situation in which the individual chooses to reduce the level of $E_c$ in order to avoid hearing the preachments of those in authority In this case the resulting prominence of $I_c$ would result in the perception of tinnitus

It is not only in the field of psychology that one finds an explanation for the lack of tinnitus in children In discussing middle ear involvement and its relation to tinnitus Thomas (1938) (49) cites the differences in the ossification of bone with age as an explanation as to why few cases of tinnitus are found in children "Bone conduction is undoubtedly a greater factor in adults than in children because of the greater density of this bone It is no doubt partly for this reason that tinnitus is uncommon in children"

The remainder of the graph in Figure 2 is best examined on the basis of sex The curve representing the incidence of tinnitus in men is quite flat after the adolescent period Except for the final decade, Figure 2 pictures man's normal working life in the labor force During these years the experiences of life are relatively equal in the type of community that Ventus (1953) (51) studied He found, and the graph demonstrates, no increase in tinnitus that might be attributed to the cumulative effects of noise trauma For the

female population there are two arresting features about the curve The sharp rise commencing about age twenty-one and ending at age fifty is the first of these Without attempting to equate the two it may be noted that this period is fairly equivalent with the period of child rearing for most women The second feature of the curve and probably the more interesting is the rapid decline in the incidence of tinnitus in the decade from fifty to sixty During this period there was a decrease of twenty cases in the population studied In the male population this period showed an increase of three Although the increase in the male incidence may not be significant from a statistical point of view *the difference between the groups of twenty three certainly is* The natural question then is what occurs to women during this period that is different from or does not occur for men? The menopause seems to be the most obvious answer What the relationship might be physiologically the author is not prepared to say On the psychological level *one might surmise that if there is a high degree of correlation between* emotional instability and onset of tinnitus the passing of the climacteric would reduce the chances of acquiring tinnitus The point here is that menopause is often the last major emotional conflict women face before old age and the loss of their spouses The problems of old age then could be the explanation for the upswing in the curve found in the sixties

If Ventus study is at all typical of mankind in general it would appear that tinnitus is directly associated with age The extent of this relationship and the characteristics of it will be known only if future cross sectional studies are conducted to supplement the data reported by Ventus

# V. MEASUREMENT OF NON-VIBRATORY TINNITUS

The first to report on a procedure for making measurements of subjective tinnitus was Josephson (1931) (29), who observed that "when a sound of the same fundamental pitch as the tinnitus is superimposed on the ear, a masking of the superimposed note by the tinnitus was found . " The method of measurement that was devised as a result of Josephson's observation is still in use today The only equipment needed is a sweep frequency audiometer with an earphone It is also desirable that the audiometer be equipped with an attenuator that is calibrated in steps of one dB To determine the pitch of the tinnitus Josephson advocated presenting two tones to the patient one well above and one well below the pitch of the tinnitus In successive steps the range between these two tones is reduced and the tone of the tinnitus is approximated The intensity is kept at a low level so that when the fundamental frequency of the tinnitus is reached the tone will be masked by it From this point on it is a matter of patient adjustment of the frequency and intensity of the tone until the exact frequency and sensation level of the tinnitus are located Josephson states that 'the threshold of the determined pitch overcoming the masking effect of the tinnitus is then determined and compared with the normal threshold of that frequency "

Minton (1923) (36) has described a method for determining the sensation level of the tinnitus in cases of unilateral involvement In this technique a tone of the same frequency as the tinnitus is applied to the opposite ear The intensity of the tone is increased until the tinnitus appears to shift to the ear receiving the tone This intensity is then assumed to be the intensity of the tinnitus

In recent years it has become apparent that patients as a group are not able to describe the tinnitus they experience in a manner that is meaningful to others Patients who have had musical training are better able to describe their tinnitus than those who have not had such training Too often tinnitus is not a pure tone but rather a mixture of complex overtones and noise elements, and the average person is unable to analyze what he is hearing

The practice of taking a more detailed account of the symptom developed in response to the poor ability of patients to describe their tinnitus Goodhill (1951) (19) saw the need for more complete description of the tinnitus as a prerequisite for scientific investigation He divided his analysis of the symptom into two parts The first part is the subjective statement of the

## CONCLUSION

After a study of the many reports about non-vibratory tinnitus, the author must agree with the statement made by Reed (1960) (41) that "unfortunately, as one reads the literature, one is left with the impression that most of what has been written is founded mainly on clinical and theoretical impressions with little basis in fact." This does not mean that some of the statements that have been made are not correct. It merely implies that data are needed before they can be accepted.

It seems obvious that increased understanding of the various physiological mechanisms that produce the symptoms of tinnitus can only come from careful study in the form of well controlled research. That more has not been done to gain this knowledge remains as much of a mystery as the symptom itself.

## SUMMARY

An examination of the literature on Tinnitus Aurium published in English has shown the wide variety of theories that have been offered concerning its causation. An equal number of methods for treating tinnitus have been employed without any showing striking success. Two studies in the past five years have shown that non-vibratory tinnitus is susceptible to accurate measurement. From recent scientific studies of the symptom, there appears some cause for hope concerning a better understanding of this perplexing problem.

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BY

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SUPPLEMENTUM 203

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# Contents

# Introduction

While the morphological development of the inner ear has been extensively studied, the functional aspects of the ontogenesis of this sense organ have attracted less attention The earliest functional studies were performed with primitive acoustic stimulation as for example clapping of the hands, striking of a tuning fork or blowing a tuned whistle The experimental animals were observed for behavioral responses such as twitching of the pinna, closure of the eyes or trunk movements Experiments of this type were performed in connection with histological studies in the rat (Kreidl & Yanase 1907, Wada 1923) and the opossum (Larsell, Mc Crady & Zimmermann 1935) An over-all estimate of the responsiveness of a developing animal to sound stimulation may be obtained in this way However, it is uncertain to what extent the results of these observations are specifically related to the development of the inner ear

Electrophysiological technique has made it possible to study the development of the inner ear function at the cochlear level The first investigations of this type were performed by McCrady, Wever & Bray (1937 1940) in the marsupial opossum The development of the cochlear microphonic potential in response to tone stimulation was studied and related to the morphological development of the organ of Corti During the progress of the present study, four investigations on the development of cochlear function have been published The cochlear microphonic and the action potential recorded from the round window in response to click stimulation, were studied in kittens by Khavina & Maruseva (1963) The development of the action potential of the auditory nerve and the cochlear microphonic potential recorded in response to click stimulation have been studied in connection with observations on the appearance of the pinna reflex and on the morphological development of the inner ear in normal mice (Alford & Ruben 1963) and in normal CBA-J mice and shaker-1 mice (Mikaelian & Ruben 1964) Schmidt & Fernandez (1963) studied the development of the endocochlear potential in the opossum, and also in a more preliminary way in the mouse and the rat The results were discussed in relation to simultaneous histological studies and the findings of McCrady *et al* (1937, 1940) on the development of the cochlear microphonic potential in the opossum

The functional development of the central auditory pathways as revealed by the cortical response evoked by click stimulation, has been studied by several investigators in most cases in connection with other studies of the ontogenesis of central nervous function (in the cat by Grossman 1955, Rose, Adrian & Santibanez 1957, Ellingson & Wilcott 1960, in the rat by Chaloupka & Myslivecek 1960, Myslivecek, Chaloupka & Springer 1961, in the rabbit and the cat by Marty, 1962) Rose *et al* (1957) in addition recorded the response at the round window and in the medial

geniculate body. Marty & Thomas (1963) found that electric stimulation of the auditory nerve fibres at the osseous spiral lamina in kittens evoked a cortical response already at birth, whereas click stimulation did not do so until later.

In the investigations referred to above the stimulating sound was applied externally to the auditory meatus. The incomplete development of the external and the middle ear may for this reason, to an unpredictable extent, have prevented the sound to reach the inner ear. This has also been pointed out by some of the investigators (Rose *et al.* 1957, Schmidt & Fernandez 1963). It is consequently uncertain, to what extent the previous work on the development of the receptor function of the inner ear may have been affected by the development of the sound conducting structures. In the present investigation this source of error was eliminated by applying the stimulating sound directly to the surgically exposed oval window by means of a closed acoustic system.

The present study was undertaken in order to demonstrate, on basis of measurements of electrophysiological signs of cochlear function in young rabbits, how the inner ear function is gradually elaborated during ontogenesis. In order to get a *more complete view of this process than the one provided by previous investigations,* it was considered advantageous to study, in the same species several of the interrelated electrophysiological expressions of inner ear function. For this purpose the cochlear microphonic potential and the summating potentials, reflecting the activity of the hair cells and the endocochlear potential, originating from stria vascularis were studied in relation to the development. A study was further made of the capacity of the developing cochlear receptors to generate propagated nerve impulses in response to tone bursts and clicks. Some observations on the appearance of the acoustic middle ear reflex will also be reported. Finally the functional observations were related to the morphological differentiation as it appears from the descriptions given in the literature and from histological studies performed in connection with the present investigation. The results presented in this paper have been briefly reported in a preliminary communication (Änggård 1964).

# Material

In selecting a suitable species for the present study the generally available laboratory animals were considered. Guinea pigs cannot easily be used since the development of the ear is completed during the prenatal period. The seasonal breeding habits of the cat impose restrictions on the continuous supply of experimental animals. Mice, rats and rabbits are easy to breed and in these species the final differentiation of the inner ear takes place after birth. For the present experimental purpose the rabbit was *found to be the most favourable species since the young mice and rats are* smaller in size and, in addition, in these species a large branch of the internal carotid artery known as the stapedial artery (*cf.* Romer 1962) crosses the middle ear passing between the crura of the stapes.

A total of 110 rabbits ranging in age from new born to 37 days were studied with

respect to one or several of the electrophysiological phenomena included in the experimental program. Histological examination of the ears was performed in 50 of these animals. In addition the histological material includes 15 animals which were not functionally tested.

Birth usually took place 30 to 31 days after conception. In case the gestational period was found to be outside these limits, the litter was not used for experiments. *The age of the animals will be given in days after birth.*

# Methods

## *Anesthesia*

The animals were anesthetized with a 20 % urethane solution which was given at a standard dosage of 0.8 ml per 100 g body weight. Half the dose was administered intramuscularly and the remaining half intraperitoneally. This was found to give a suitable rate of induction of anesthesia. Smaller additions amounting to 10-20 % of the initial dose were sometimes given intramuscularly. This produced a satisfactory level of anesthesia characterized by regular respiration and absence of spontaneous movements. The temperature of the animal was followed by means of a mercury thermometer introduced subcutaneously in the groin. The body temperature was kept at 37° ± 1° C with an infrared lamp.

## *Surgical Procedures*

The animal was tracheotomized and a polyethylene tube inserted in the trachea. The skull was exposed by resecting a flap of skin beginning at the occiput and proceeding anteriorly along the sagittal suture to the snout. This allowed the head to be fixed in the stereotactic head holder which will be further described below. The animal was placed with its left side up on a small platform to which the head holder was clamped. This arrangement provided free access to the left ear and the adjacent parts of the skull. The platform was mounted to a heavy base of cast iron by means of a ball and socket joint. This permitted the platform to be tilted as required in order to provide a suitable orientation of the head of the animal.

The left ear which was routinely used for the experiments was surgically prepared under a dissection microscope (Zeiss Epitechnoscope). After a wide skin incision the posterior part of the mandible was resected and the soft tissue retracted to expose the external auditory meatus and the bulla. The pinna was amputated. The posterior auricular artery was ligated and cut together with the facial nerve. Subsequently the styloid and the paraoccipital processes were resected and the muscles attached to them retracted to provide a wider exposure of the bulla. The external auditory meatus the tympanic membrane and any remaining embryonal mesenchymal tissue found in the middle ear were carefully removed. The posterior and

anterior portions of the tympanic ring were resected leaving the middle portion which is situated anteriorly to the canal of the internal carotid artery After extirpation of the malleus and the incus the tendon of the stapedius muscle was cut The crura of the stapes were removed leaving the footplate intact To provide enough space around the oval window for the application of the stimulating device the facial nerve was removed from its recess and adjacent bony ridges trimmed down The exposure allowed wide access to the medial wall of the cochlea the oval and the round windows and the adjacent parts of the occiput By careful dissection these procedures could be performed with only minor haemorrhage Slight bleeding from the cut surfaces of the vascularized developing bone was controlled by the application of bone wax

In experiments involving measurements in scala media the spiral ligament was exposed by carefully trimming down the covering bone with the aid of the pointed edge of a disposable surgical blade until only a thin lamina remained The periphery of the lamina was cautiously transected and the central portion lifted away The animal was not used for experimentation when obstructed circulation or bleeding of the vessels in the spiral ligament were observed

## *Stimulation*

The sound was applied by means of a closed acoustic system which was sealed over the oval window The dimensions and construction of the stimulating device is shown in Fig 1 The electric signals were fed to a one inch condenser microphone (Bruel & Kjaer type 4131) which thus served as an electro acoustic transducer This was connected as shown to a rigid and transparent plastic tube in the centre

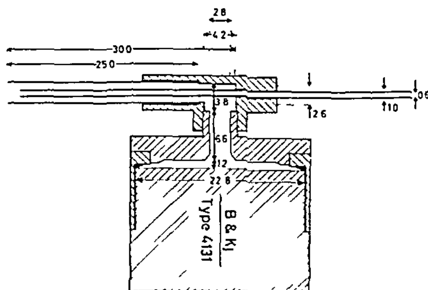



Fig 1 Schematic ... of the stimulating device Dimensions in millimeters

of which a probe tube was situated The terminal orifice of the probe tube was located 1 5 mm from the peripheral end of the plastic tube

The acoustic probe was connected by means of a short polyethylene tube and a 1 mm bore adapter (from probe kit, Bruel & Kjaer, type UA 0040) to a half inch condenser microphone (Bruel & Kjaer, type 4134) with cathode follower (Bruel & Kjaer, type 2615) To minimize standing waves in the plastic tube a piece of woolen yarn was introduced in the proximal 15 mm of the tube The wool had been treated with a water repellent to prevent it from absorbing moisture

The stimulating device was mounted with a ball and socket joint to a micro manipulator (Prior & Co Ltd ), which allowed movements along three right angled coordinates The peripheral orifice of the plastic tube was adjusted into position over the oval window and sealed to its margin with vaseline The transparency of the plastic tube made it possible to ascertain that it fitted tightly to the margin of the oval window and that no accumulation of fluid occurred in the tube

The arrangement of the electronic equipment connected to the stimulating device is shown schematically in Fig 2 The condenser microphone serving as an electro-acoustic transducer had a polarization voltage of 240 V Continuous sinusoidal signals were provided by the built in tone generator of the wave analyzer (Radiometer,

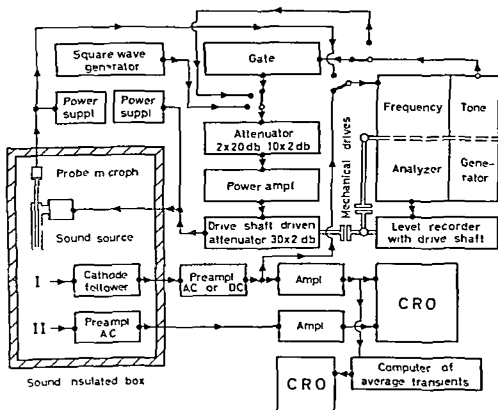



Fig 2 Block diagram showing arrangement of stimulating and recording apparatus

type FRA 2 T). The intensity of the signal could be adjusted over a 60 db range divided in 2 db steps by a manually operated attenuator. After having passed through a power amplifier the signal could be attenuated over an additional 60 db range also divided in 2 db steps with another attenuator, which could be rotated by means of a connection to the drive shaft of the level recorder (Brüel & Kjaer, type 2305). A particular feature of the combined tone generator and wave analyzer was that the frequency of the generated signal always remained the same as the one to which the wave analyzer was tuned. The frequency control of the instrument could be connected to the drive shaft of the level recorder for measurements of the frequency characteristic of the stimulating device.

The sound pressure at the oval window was determined with the calibrated probe microphone connected to the wave analyzer, the output of which was recorded on the level recorder. The wave analyzer functioned as a selective voltmeter with a bandwidth of 25 cps. The sound pressure determinations were made at the

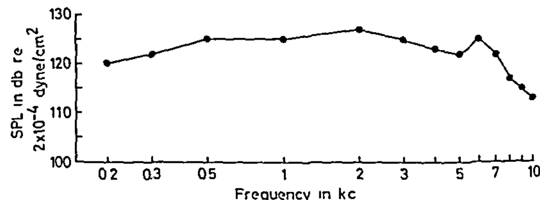



Fig. 3. Frequency characteristic of stimulating device at maximum sound intensities employed. Mean values from probe microphone measurements in ten consecutive experiments. Individual values were within ± 1 db from 0.2 to 5 kc and within ± 2 db from 6 to 10 kc.

maximum sound intensity over a frequency range of 0.2 to 10 kc. The values obtained in each experiment were employed in the calculation of the results of the *electrophysiological measurements made in that particular experiment*. In the following account *the sound intensity will be given in db relative to* $2\times10^{-4}$ *dyne/cm*$^2$ unless otherwise stated. The mean values of the sound pressures obtained in ten consecutive experiments are shown in Fig. 3. The values varied within ± 1 db from 0.2 to 5 kc and within ± 2 db from 6 to 10 kc. Observations at intervening frequencies showed that no pronounced resonance peaks were present.

Controls were made to ascertain that the sound intensity at the fundamental frequency increased linearly in relation to the electric signal input to the electro-acoustic transducer. This was found to be the case even in the frequency range below 1 kc where distortion of wave form was observed at high intensities. This distortion was found almost exclusively to be due to the presence of second harmonics. The percentage of second harmonics at 0.2 kc, 0.5 kc, 1 kc and 3 kc is shown in Fig. 4.

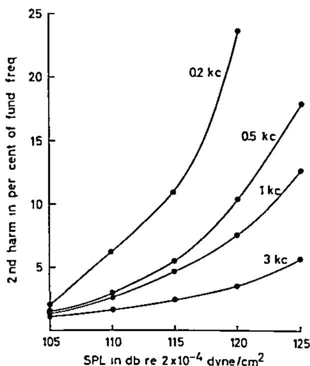



Fig 4 Second harmonic content of stimulating sound at indicated frequencies in relation to sound intensity at the fundamental frequency

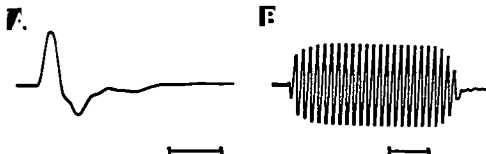



Fig 5 Condenser microphone records of signals used as standard stimuli See text A rarefaction click Time bar 0 5 msec B 3 kc tone burst at 125 db Time bar 2 msec Same amplification in both records

For certain stimulation purposes rarefaction clicks were produced by activating the electro acoustic transducer with a square wave having a duration of 0 12 msec. Grass Instruments Co, square wave stimulator type S 4 C) An estimate of the shape of this acoustic transient was obtained in the following way The peripheral orifice of the plastic tube of the stimulating device was connected to the measuring microphone by means of a suitable adapter (Brüel & Kjaer, probe kit type

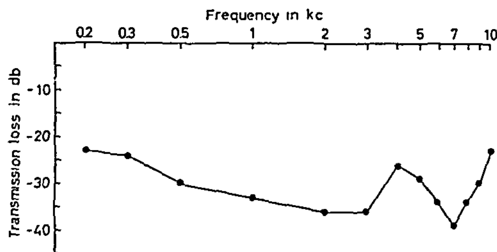



Fig 6 Transmission loss after removal of the middle ear in 36-day rabbit The curve represents the difference between the sound intensities required at the tympanic membrane and those required at the exposed footplate of the stapes in order to produce a cochlear microphonic potential of 2 $\mu$V r m s

0010) so that the orifice was situated 1 mm above the microphone diaphragm The output of the microphone was displayed on a cathode ray oscilloscope (Tektronix, type 502) A record obtained in this way is shown in Fig 5 A

Tone bursts of variable duration were obtained by passing the signal from the tone generator through an electronic gate (Moller 1961) Usually a standard tone burst with a frequency of 3 kc an intensity of 125 db and a duration of 8 msec was used The record shown in Fig 5 B was obtained in the manner described in the preceding paragraph As shown the amplitude rises to 90 per cent of the maximum value in about 1 msec At the end of the tone burst the amplitude declines at the same rate

The reduction of sound transmission produced by removal of the middle ear structures has been studied by many previous investigators under different experimental conditions To determine the transmission loss existing under the specific conditions imposed by the technique employed in the present investigation the following experiment was performed Leaving the middle ear intact, the stimulating d[illegible] with its probe microphone was connected to the external auditory meatus The [illegible] pressure required to produce 2 $\mu$V r m s of cochlear microphonic potential fro[illegible] the round window was determined The same procedure was repeated with stimulati[illegible] the stapes footplate The difference between the values obtained under the different stimulation conditions are shown in Fig 6 The curve is similar to the [illegible] presented by Wever Lawrence, & Smith (1948) in experiments in cats with the [illegible] technique

Care w[illegible] ta[illegible] that the noise in the laboratory did not interfere with measurem[illegible] level wi[illegible]n a bandwidth of 25 cps was determined over a frequency [illegible] 0 1 [illegible] 12 kc The n[illegible]se level at 0 1 kc was found to be 35 db

At higher frequencies it decreased continuously to a level of 15 db at 12 kc It thus appears that in the high frequency range the absence of the middle ear offers full protection against the noise However, in the frequency range below 0 5 kc the attenuation is only about 25 db leaving a residual 10 db that might enter the inner ear This residual noise could be eliminated by a sound insulated box, in which the animal was kept during the experiment The box provided a sound insulation of 15 to 20 db in the low frequency range and about 40 to 50 db in the upper frequency range In practice however it was found that no appreciable influence on the electro physiological measurements could be observed if the door of the box was left open For this reason the noise protection of the box was usually not employed

## Recording

The arrangement of the recording equipment is shown in Fig 2 The *cochlear microphonic potential* was recorded with Ag AgCl electrodes The activity at the round window was recorded against a reference electrode placed in contact with the neck muscles Fluid and mesenchymal connective tissue at the round window was carefully removed The electrodes were connected to the cathode follower of an AC coupled preamplifier (Grass Instruments Co, type P 6), the output of which was recorded either on a cathode ray oscilloscope (Tektronix, type 502), or after having passed through the wave analyzer, operated at a bandwidth of 25 cps on the logarith mic level recorder Due to the frequency selectivity of the wave analyzer the noise level of the recording system was below 0 5 $\mu$V r m s The frequency response of the recording equipment was linear within the employed frequency range

Since the drive shaft of the level recorder could be mechanically coupled to one of the 60 db attenuators incorporated in the circuit supplying the electro-acoustic transducer the magnitude of the cochlear microphonic potential in relation to in crease of the sound intensity at any particular frequency could be automatically re corded By selecting a suitable drive shaft speed this recording procedure could be performed in 20 seconds The arrangement prevented unduly long experimental sessions during which the condition of the experimental animal could have deterio- rated In addition the analysis of the experimental data was greatly facilitated

In those experiments in which the P 6 preamplifier was used for the recording of action potentials in the cochlear nuclear complex a second recording channel with an AC preamplifier (Grass Instruments Co type P 8) was provided for simul taneous recording of the cochlear microphonic potential from the round window

The *endocochlear potential* was measured with glass capillary microelectrodes pro- duced as described by Haapanen Kolmodin & Skoglund (1958) Pyrex glass with an outer diameter of 0 8—1 mm and an inner diameter of 0 5—0 6 mm was em ployed The microelectrodes were filled with 2 7 M KCl by boiling in ethanol at reduced pressure followed by transfer into distilled water and subsequently into the KCl solution (Tasaki Polley & Orrego 1954)

Before use the impedance of the electrodes was estimated in the following way

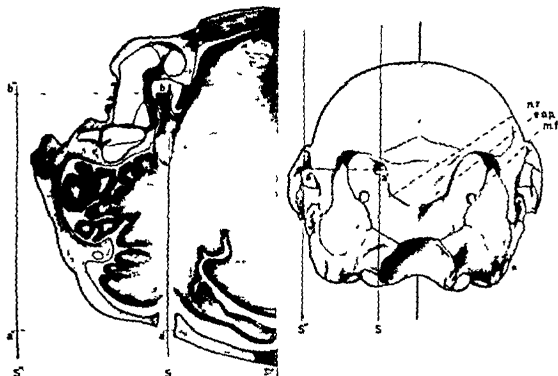



Fig 7 Transverse section of rabbit skull along an electrode track S' sagittal plane through the electrode track a' centre of hole drilled in the occiput b' target area in the auditory nerve, S" sagittal plane immediately lateral to the stylomastoid process, a", intersection between S" and the perpendicular through a' b" intersection between S" and the perpendicular through b' and the center of the footplate of the stapes

Fig 8 Posterior view of skull of young rabbit N r nuchal ridge, e o p external occipital protuberance m f mastoid foramen See also Fig 7

The electrode was mounted in a micromanipulator (Prior & Co Ltd ) and connected to the input of the cathode ray oscilloscope and the tip immersed in a shallow vessel containing Ringer solution A square wave was introduced between ground and the Ringer solution and the reduction of the signal due to the voltage division between the electrode impedance and the input impedance of the cathode ray oscilloscope (1 megohm), was observed The tip of the electrode was broken against the bottom of the vessel until the impedance was found to be about 5 to 10 megohm

Subsequently the microelectrode was connected to the cathode follower of the DC coupled preamplifier (Grass Instruments Co type P 6) by means of an Ag AgCl electrode A similar grounded electrode with an agar KCl bridge placed in contact with the neck muscles served as reference In some experiments in which long time stability was particularly critical as for instance in the experiments on the effect of asphyxia on the endocochlear potential, calomel cells with agar-KCl bridges were used The grid current of the cathode follower input was of the order of $10^{-11}$ A

When the microelectrode was used also for recording of the *cochlear microphonic potential* and the *summating potentials* in scala media the time

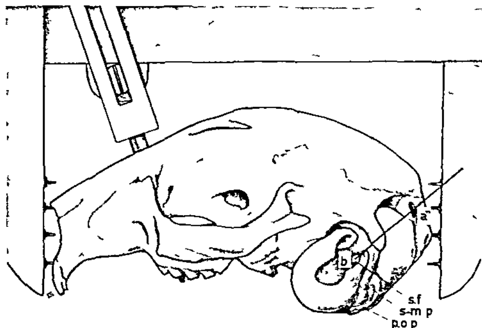



Fig 9 Lateral view showing skull of young rabbit in stereotactic head holder s f, stapes foot plate, s m p, stylomastoid process, p o p, paraoccipital process See also Fig 7

constant of the recording equipment and the electrode was tested by observing the response to a square wave applied to the animal. The tip was broken against the bone of the otic capsule until the time constant was below 50 $\mu$sec, corresponding to an upper cut-off frequency of 3 2 kc. The low frequency cut-off of the recording system was 2 cps

For recording of *electric responses in the cochlear nuclear complex* a stereotactic technique for insertion of the electrode was devised The skull of the animal was rigidly fixed in a headholder (Fig 7) made out of a sliding caliper the jaws of which had been provided with short points offering a secure hold at the midline of the occiput and between the anterior incisor teeth A third point of attachment was established by inserting a needle through the center of the naso-frontal suture, down through the hard palate The needle was fixed to the head holder with an adjustable attachment The head holder was clamped to a pair of bars on the platform on which the animal was lying with its left side facing upwards The plane of the head holder, representing the sagittal plane of the animal, was adjusted, with the guidance of a water-level, into the horizontal plane by means of the ball and socket joint on which the platform was mounted

The stereotactic electrode was a stainless steel wire 0 05 mm in diameter, insulated and rendered rigid by pulling a Pyrex glass coating over it in the apparatus used for the production of glass microelectrodes The glass tip was broken leaving about 0 2 mm length of wire uncovered The electrode was mounted on a rectangular

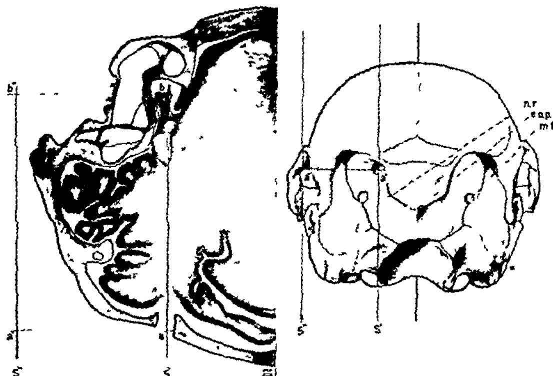



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When the microelectrode was used also for recording of the *cochlear microphonic potential* and the *summating potentials* in scala media, the time

subsequent histological procedure was the same as the one described below, except that the specimens were sectioned in a plane parallel to the electrode track Every fifth section was stained and mounted With the second type of fixation the resulting prussian blue deposits were weaker In these cases only the course of the electrode track could be determined

## *Histological Procedures*

The specimens used for study of the morphological development of the inner ear were fixed with Heidenhain Susa solution The fixative was administered by perfusion through the aorta after having washed out the blood with Ringer solution Equally good results were obtained by flooding the cochlea with the fixative through the opened oval and round windows After hemisection of the head and removal of the part situated anteriorly to the temporal bone the specimens were immersed in the fixative over night They were subsequently decalcified in a 5% solution of trichloroacetic acid dehydrated with increasing concentrations of ethanol and embedded in celloidin The blocks were sectioned in a plane parallel to the modiolus The sections (about 15 μ thick) were stained with hematoxylin-eosin and mounted in canada balsam

# I. Cochlear Microphonic Potential

It is now generally recognized that the cochlear microphonic potential (CM) is generated by the hair cells of the organ of Corti (*cf.* Wever & Lawrence 1954, Davis 1957 and 1960). Evidence has been obtained suggesting that CM originates specifically from the external hair cells (Davis *et al.* 1958 b).

Although the nature of the events leading to excitation of the afferent auditory fibres is not yet fully known, CM together with the summating potentials (SP) are considered to be the receptor potentials of the organ of Corti (Davis 1957, 1961). They thus represent the first step in the processes leading to initiation of auditory nerve impulses and, consequently, are of primary interest in a study of the ontogenetic development of cochlear function. The first section of this paper will report an investigation, undertaken in order to study the early appearance of cochlear receptor function and its later development as revealed by the capacity of the hair cells to respond to acoustic stimulation by generation of CM. A corresponding investigation on SP will be reported in Section II below.

Recording from the round window the intensity function of CM, *i.e.* the logarithmic value of the voltage of CM in relation to increasing sound intensity (in db relative to $2 \times 10^{-4}$ dyne/cm$^2$), was studied at selected standard frequencies between 0.2 and 10 kc (0.2 kc, 0.5 kc, 1 kc, 2 kc, 3 kc, 5 kc, 7 kc, 9 kc, 10 kc). Since the intensity function of CM is continuously graded, a true threshold cannot be established. For this reason an arbitrary threshold was selected, representing the sound intensity at which CM attained 2 $\mu$V r.m.s. CM usually increases as a simple linear logarithmic function of sound intensity. However, at a certain intensity level, the intensity function of CM deviates from the linear course and subsequently passes through a maximum. In the present investigation the sound intensity at which the intensity function of CM deviated by 2 db was determined as well as the magnitude of CM at this intensity. No attempts were made to record maximum CM, since the sound intensity required would have involved a risk of injury to the organ of Corti due to overstimulation.

In connection with the studies on SP, CM was also recorded from scala media with glass microelectrodes.

## *Results*

Before the animals were 4 to 5 days old, no CM above 2 $\mu$V r.m.s. could be recorded from the round window, when using the maximum output of the sound source (see Fig. 3) at frequencies between 0.2 kc and 10 kc. This observation was made in experiments on one new born, two 3-day and eight 4 day animals. In two other 4-

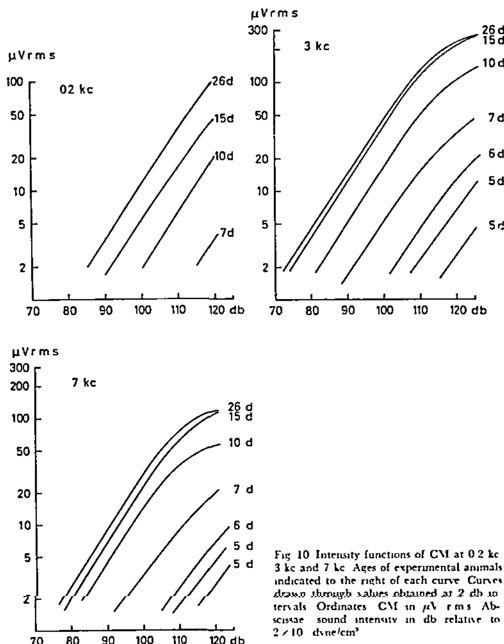



Fig 10 Intensity functions of CM at 0 2 kc 3 kc and 7 kc Ages of experimental animals indicated to the right of each curve Curves drawn through values obtained at 2 db intervals Ordinates CM in μV r m s Abscissae sound intensity in db relative to 2 × 10 dyne/cm²

day animals CM, somewhat above the 2 μV r m s level could be recorded within the narrow frequency range of 2 to 4 kc In one of the three 5-day animals tested, the magnitude of CM and the frequency range in which it could be observed slightly exceeded that of the two 4-day animals Records from the two other 5-day animals are included in Fig 10 and 11

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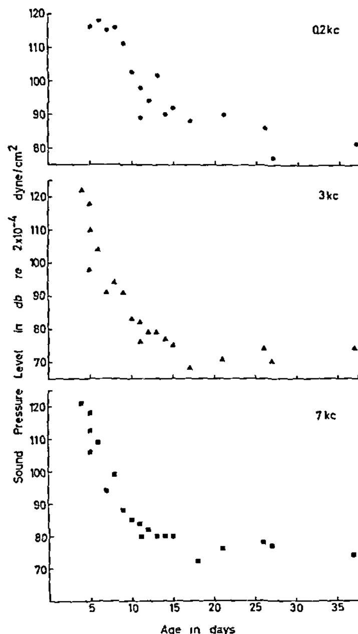



Fig 12 Development of arbitrary threshold of CM at frequencies indicated in respective diagrams Ordinates sound intensity required to produce CM of 2 $\mu$V r.m.s Abscissae age of experimental animals

threshold decrease described above is taking place At frequencies below 1 kc values could not be obtained because in the majority of the young animals the maximum sound pressure available from the sound source was not sufficient to attain the intensity range where nonlinearity of the intensity function of CM ensued

During development there is a decrease of the sound intensity at which 2 db departure from linearity was observed at 3 kc and 7 kc (Fig 14) This trend is further emphasized by the observation that in one 4-day and in two 5 day animals the

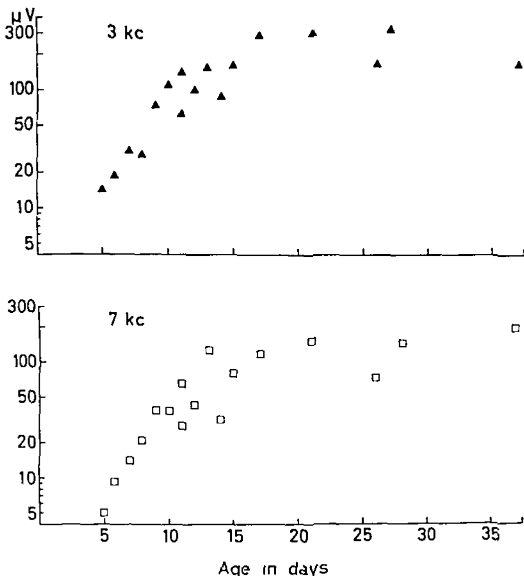



Fig 13 Development of magnitude of CM at 2 db departure from the linear course of the intensity function of CM Frequency of stimulating sound indicated in respective diagrams Ordinates magnitude of CM in $\mu$V r m s Abscissae age of experimental animals

sound intensity at which nonlinearity ensued was higher than the maximally available one, since the intensity function of CM had a linear course below this level

The changes described above (Fig 12 13 and 14) imply that during the progress of development the intensity function of CM is displaced upwards and to the left in the intensity voltage diagram (cf Fig 10)

*Attempts were also made to record CM with an electrode introduced into a small* hole drilled in the apex of the cochlea However, frequent failures to obtain stable recording conditions made the records difficult to interpret

In connection with the experiments on SP (see Section II) CM was also recorded

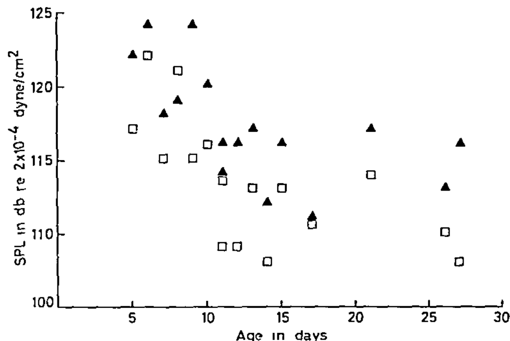



Fig 14 Sound intensities required at different stages during development in order to produce 2 db departure from the linear course of the intensity function of CM at 3 kc (filled triangles) and 7 kc (open squares)

with glass microelectrodes, introduced into scala media through the spiral ligament 2 to 3 mm from the basal end of the endocochlear duct The response to stimulation with short tone bursts at 3 kc was selected for study, since the electrodes showed a high frequency cut-off at 3 2 kc and at frequencies below 1 kc distortion was observed at high intensities Comparison of the magnitude of CM recorded from scala media in the youngest animal (5 days old) with that recorded in the two oldest (15 and 19 days old) when stimulating at a sound intensity within the linear range of the intensity function of CM showed that the difference was of the order of only 10 db This is in contrast to the corresponding difference in magnitude of CM recorded from the round window, which amounts to more than 20 db when the most advanced 5-day and the 6-day animals are compared with the older animals (Fig 11) This discrepancy will be further discussed below

## *Discussion*

In experiments on the guinea pig cochlea von Bekesy (1951 a) has shown that the bony otic capsule provides electric insulation of the inner ear and that the resistance to the body is mainly formed by the vessels and the nerves entering the cochlea Whether the same applies to the developing cochlea is not known However, since the

otic capsule is composed of richly vascularized developing bone during the early stages of the development, it can be assumed that the resistance between the cochlea and the body is lower in the younger than in the older animals In the former the shunting effect may consequently reduce the voltage picked up from the round window to a larger extent than in the latter, in which ossification is more complete

It was further shown by von Bekesy (1951 a) that the cochlear canal forms an electric transmission line divided lengthwise by the low resistive cochlear partition Along this conductive system a voltage, applied between electrodes on either side of the cochlear partition, attenuates about 6 db per millimeter distance from the voltage source Other observations (Tasaki, Davis & Legouix 1952 Tasaki & Fernandez 1952, Tasaki 1957, Misrahy, Hildreth, Shinabarger & Gannon 1958) on the local spread of CM as recorded with differential electrodes placed on opposite sides of the cochlear partion support this view and indicate that CM recorded with a single electrode on the round window originates almost exclusively from hair cells in the lower part of the basal turn Simmons & Beatty (1962) in experiments on the effect of overstimulation of the cochlea in cats obtained evidence supporting this conclusion They found that the best correlation between the auditory loss (as revealed by conditioning tests) and CM at the round window was found in animals in which the frequencies from 16 kc and above were affected whereas an extensive loss below this range had only a minor effect on CM recorded from the round window According to Wever & Lawrence (1954) CM recorded at the round window originates from a more extensively distributed hair cell population However, there is reason to believe that their estimation of the attenuation along the cochlear duct was too low (*cf* Tasaki 1957)

Whether the origin of CM recorded from the round window in developing animals is restricted as in adults, to the lower part of the basal turn is uncertain, since the assumed low electric insulation provided by the developing bone in the partitions separating adjacent turns may result in a larger contribution from more apically situated hair cells in the young animals than in adults On the other hand if the conductivity of the cochlear partition is higher in the developing animal than in the adult the attenuation along the cochlear duct would be higher in the former The contribution from apically situated hair cells would consequently be even less in developing animals than in adults However it seems reasonable to assume that the CM activity recorded from the round window in developing animals mainly derives from the hair cells of the lower part of the basal turn since these are closest to the recording site

Since histological observations (see Section VI) indicate that the development of the organ of Corti starts in the basal turn and proceeds apically an electrode placed at the round window is in a favourable position to record early CM activity Still more advantageous recording conditions are achieved by recording from scala media where CM has a larger magnitude than in the corresponding section of the peri lymphatic space (von Bekesy 1952 b Tasaki Davis & Legouix 1954) It has been shown by von Bekesy (1952 b) that this recording site is also more selective in fa vour of hair cells close to the electrode than an electrode in contact with the

perilymphatic space However, records obtained with the two different techniques are not directly comparable, since the recorded activity does not originate from the same hair cell groups and may in addition be differentially influenced by developmental changes of the conductivity of the cochlea

In evaluating the results of the measurements of CM in the low frequency range, it is necessary to consider that frequency synchronized activity from the auditory nerve may to an unknown extent, have added to CM recorded in response to frequencies at least below about 1 kc (*cf* Stevens & Davis 1938) It has been shown (Stevens & Davis 1936) in records from the auditory nerve in guinea pigs that the frequency synchronized activity is pronounced up to a frequency of about 800 cps and decreases by steps at certain frequency intervals until at about 3 kc it is hardly noticeable The decrease of the threshold at 0 2 kc may thus in the present experiments have been affected by gradually appearing contributions of frequency synchronized activity from the auditory nerve On the other hand it cannot be excluded that the different time course of development at 0 2 kc may have been due to later appearance of activity from more apically located hair cells during the gradual extension of the development of the organ of Corti in the apical direction (see p 17)

Wever Bray & Lawrence (1940 a b c and 1941) and Wever & Lawrence (1941 1951) have performed experiments designed to establish the locus of the mechanism responsible for the nonlinear deviation of the intensity function of CM From these investigations it was concluded that this phenomenon does not arise in the middle ear, nor in the cochlear fluids but in the organ of Corti Two different mechanisms were considered to account for this feature of the intensity function *i.e.* one by which the acoustic energy is mechanically transformed into harmonic distortion products and a second one connected with the mechanoelectrical transduction in the hair cells The former mechanism was considered to be acting in the intensity range where the intensity function starts to deviate from the linear course and the second one in the part of the curve, where it passes through a maximum as a result of overloading Davis & Eldredge (1959) found that when CM is recorded with differential electrodes the nonlinearity of the intensity function of CM at frequencies above about 4 kc appears without noticeable distortion of wave form As a corollary to their hypothesis on the production of SP (see p 27) they suggested that the nonlinearity is caused by a one way shift of the tectorial membrane resulting in restriction of the transverse movements of the organ of Corti From the account given above it appears that there is so far no conclusive evidence as to what parts of the organ of Corti are engaged in the mechanism governing the nonlinearity of the intensity function of CM However, the parameters by which this function has been studied in the present investigation represent different aspects of the process by which acoustic stimulation is converted into an electric response from the hair cells

In the present investigation it was observed that CM could not be recorded from the round window or from the scala media until the animal had attained an age of 5 or occasionally 4 days this suggests that the hair cells of the basal turn of the cochlea have not begun to respond to sound stimulation before this age Since all

otic capsule is composed of richly vascularized developing bone during the early stages of the development, it can be assumed that the resistance between the cochlea and the body is lower in the younger than in the older animals In the former the shunting effect may consequently reduce the voltage picked up from the round window to a larger extent than in the latter, in which ossification is more complete

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parameters of the intensity function of CM at an age of 15 to 20 days after birth attain values, which are subsequently maintained at the same level, it is likely that the development is completed at that age

As demonstrated, the intensity function of CM, plotted in a diagram of the type shown in Fig 10, is displaced upwards and to the left during the development This is in agreement with observations by McCrady, Wever & Bray (1937) in similar experiments in the opossum However, because of the stimulation technique employed by these investigators (*cf* p 6) the displacement along the abscissa observed by them must probably partly be attributed to a gradually increased sound transmission through the external and middle ear In connection with studies on the development of the endocochlear potential in the opossum, Schmidt & Fernandez (1963) actually observed the auditory meatus to be closed up to the age at which McCrady *et al* had found CM to appear In the present investigation the sound stimulus was applied at the stapes footplate, which could be observed through the dissection microscope to have a normal mobility throughout the developmental period studied, this suggests that the displacement along the abscissa is due to functional changes within the cochlea Irrespective of the nature of the process in the organ of Corti responsible for the nonlinearity of the intensity function of CM it seems improbable that the immature hair cells would be able to respond linearly at an intensity range, where the fully developed hair cells respond nonlinearly It seems more likely that, as development proceeds, the acoustic energy is transmitted with gradually increasing efficiency to the site where nonlinearity arises

The displacement of the intensity function of CM along the ordinate is presumably partly due to reduced shunting of CM as a result of gradually increasing electric resistance of the otic capsule during the course of its ossification However, this displacement is probably also the result of successively increased sensitivity implying that, as development proceeds, each hair cell responds with an increasingly larger electric signal to a given stimulation It is also possible that there is a successively increasing number of hair cells contributing to the round window response, as a result of the gradual extension of the development of the hair cells towards the apex and in the transverse dimension of the organ of Corti (see Section VI) It is very likely that the gradual increase of the endocochlear potential (see Section III) contributes to the augmented sensitivity of the hair cells

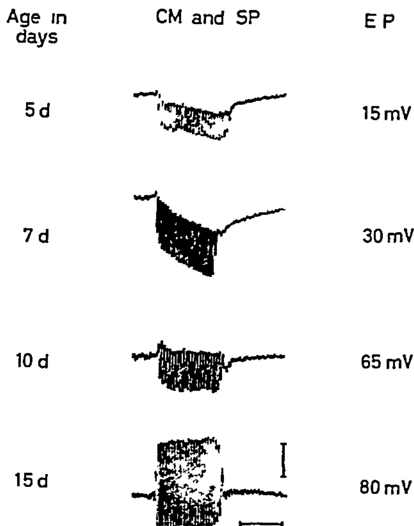



Fig 15 CM and SP recorded from scala media of the basal turn at ages indicated to the left. The endocochlear potential recorded in these animals indicated to the right. Stimulation standard 3 kc tone burst at 125 db. Downward deflection indicates negativity in scala media. Horizontal bar 5 msec. Vertical bar 0 5 mV Same amplification in all records

age of 5 days In records of SP from scala media in a 5-day animal (Fig 15) an initial rapid negative DC shift was observed followed by an additional slower increase of the negativity After termination of the stimulation the potential returned towards baseline in the same sequence At the end of the 8 msec long tone burst SP had not yet attained its maximum magnitude Fig 16 shows a DC record obtained from an experiment on a 6-day animal in response to a 3 kc tone with a duration of about 150 msec As seen the maximum magnitude of SP was attained after about 60 msec

a difference in longitudinal stretch between the tectorial membrane and the rest of the organ of Corti during propagation of the traveling wave Another mechanism, discussed in less detail, was postulated to produce a transverse one way shift of the tectorial membrane towards the limbus, resulting in selective stimulation of the external hair cells to produce SP+

An alternative type of mechanism to account for the production of SP emerges from recent observations by Flock on the lateral line organ in fish (1965) He confirmed the previous observation by Kuiper (1956) that the lateral line organ responds to vibratory stimulation with a negative DC shift, on which the characteristic microphonic response is superimposed It was further clearly demonstrated that this DC response is not caused by a steady one way displacement of the sensory hairs He also showed that the input-output transfer function of the hair cells was non linear and suggested that the negative DC potential is produced by a cellular integrating mechanism summating the alternating microphonic potential over time

Summating potentials have also been recorded from the avian cochlea (Schmidt & Fernandez 1962, Stopp & Whitfield 1964) Ross & Whitfield (1965) assumed that the basilar membrane responds nonlinearly to a symmetrical driving force Due to phase differences between the output from individual hair cells due to propagation of the traveling wave, it was suggested that alternating signals would tend to cancel, whereas a DC shift due to any nonlinearity would be unaffected

SP has previously not been studied during the development of the organ of Corti Since information in this respect would provide a wider basis for the evaluation of the development of the receptor function of the inner ear, the experiments presented below were performed

## Results

Some of the results presented in this section were obtained from a group of 10 animals ranging in age between 4 and 19 days For the recording of SP and EP a glass microelectrode was inserted through the spiral ligament 2 to 3 mm from the basal end of the cochlear duct The recording conditions were stable and no appreciable reduction of SP or EP could be observed during experiments lasting one to two hours In addition confirmatory evidence was provided by observations on SP recorded from the round window in the majority of the animals investigated with respect to CM from the round window (see Section I) and the electric response in the cochlear nuclear complex (see Section V)

SP was usually studied in response to the standard 3 kc tone burst at 125 db (see Fig 5 B), since frequent checks at lower and higher frequencies showed that SP was most prominent around this frequency Unless otherwise stated SP was recorded with AC coupling giving a low frequency cut off at ? cps In the records of SP, downward deflection indicates negativity at the recording electrode relative to a grounded reference electrode in contact with the neck muscles

SP could not be recorded from scala media or from the round window before the




g 18 CM and SP recorded from the round window in 5 day (A) and 15 day (B) rabbits imulation standard 3 kc tone burst at 125 db Downward deflection indicates negativity at e round window Horizontal bar 5 msec Vertical bar 50 $\mu$V (A) and 100 $\mu$V (B)

Under the influence of asphyxia, established by intraperitoneal administration of neuromuscular blocking agent (Flaxedil May & Baker Ltd) in a dosage sufficient produce respiratory paralysis the early type of SP was found to decrease gradually magnitude (Fig 17) without any temporary change of polarity or increase in magni-de

The subsequent course of the development of SP appears from the records of Fig 5 During the 2 days following the appearance of SP the negativity of SP in eases in magnitude However, as seen in the record from the 10 day animal the egativity later becomes smaller and finally in a 15 day animal SP is found to be ositive

The effect of asphyxia on SP in a 15 day animal (Fig 17) is different from that bserved at an age of 5 days At the beginning of asphyxia the positive SP changed nto a negative one which was still prominent at the time when CM was only iscernible as a faint ripple The onset and the return to baseline of this negative P had a slow exponential course without any rapid component at the beginning nd at the end of the stimulation like the SP observed during the early period of evelopment

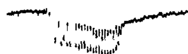
The SP recorded at the round window was always of a smaller magnitude than ne one recorded from scala media The DC shift observed at the round window at n age of 5 days was positive (Fig 18A) corresponding to the negative SP recorded scala media since the former recording site was situated on the opposite side of ne generators in the organ of Corti For the same reason the round window DC esponse in a 15 day animal was slightly negative (Fig 18 B)



Fig 16 CM and SP recorded from scala media of the basal turn in 6 day rabbit DC recording Stimulation tone burst with a frequency of 3 kc, intensity of 125 db and duration of 150 msec Negativity downwards Horizontal bar 25 msec Vertical bar 0 5 mV

5 days

15 days



0 min





3 min





10 min





18 min



Fig 17 Effect of asphyxia on CM and SP recorded from scala media of the basal turn in rabbits 5 and 15 days old Duration of asphyxia indicated in the center Stimulation standard 3 kc tone burst at 125 db Negativity downwards Horizontal bar 5 msec Vertical bar 0 5 mV Amplification increased 2 5 × in the two lowest records from the 5 day animal

response until finally SP+ would dominate to give a positive response This interpretation is supported by the observations on the effect of asphyxia on SP at the early and at the final stages of the functional development of the cochlea

It seems improbable that the reversal of the polarity of SP observed during the development would be produced by a change in the mechanical bias acting on the cilia of the hair cells During the development the difference in increase of the size of the supporting structures of the organ of Corti results in a tilting of the position of the reticular membrane from a plane parallel with the basilar membrane into a final position in which the reticular membrane inclines towards the modiolus (Retzius 1884 Held 1909) In guinea pigs injection of an inert solution into scala tympani results in a change of the position of the reticular membrane in relation to the tectorial membrane in the same direction This produces reversal of an originally negative SP into a positive one (Davis *et al* 1958 a) However it seems unlikely that the slow positional changes occuring during development in a similar way would result in a maintained mechanical bias

The observations on the development of SP indicate that the receptor function of the organ of Corti starts to appear at an age of 5 days and that the development is completed at least in the basal turn at about the same time as that indicated by the development of CM *i e* at an age of about 15 days

## *Discussion*

The observation that SP as recorded in the present investigation was positive at 3 kc is in contrast with what is usually found in the guinea pig, in which SP is negative in the lower part of the basal turn in response to frequencies above 2 to 4 kc and positive usually only at a frequency as low as 0.5 kc (Davis *et al.* 1958 a). A possible explanation for this dicrepancy could be that the resonance curves of the basilar membrane are different in these two species. According to Davis *et al.* a negative SP *is recorded close to the maximum of the traveling wave envelope*, whereas a positive SP appears further down on the basal slope of the envelope. The resonance curve of the basilar membrane of the rabbit is not known. However, it might be assumed that the lower part of the basilar membrane of the rabbit, as for example that of the rat (von Békésy 1944), is tuned to higher frequencies than in the guinea pig. The maximum of the traveling wave envelopes at those frequencies, at which SP is negative in the guinea pig, would then in the rabbit be situated further apically, and the recording electrode would consequently be in closer proximity to the basal slopes of the envelopes. A positive SP would then be expected to be recorded in response to higher frequencies in the rabbit than in the guinea pig.

As mentioned previously (p. 27) other investigators using guinea pigs observed a change from a negative SP to a positive one as a transitory initial effect of anoxia. In the experiments published by Konishi *et al.* (1961) this reversal of polarity occurred, when the anoxia was so severe that EP had been considerably reduced. It is unlikely that the positive SP recorded in the mature animals would have been due to inadvertent surgical trauma as EP in these experiments had a normal magnitude (+80 mV). Furthermore SP had the same polarity when recorded from the round window *in which case the inner ear had not been subjected to surgery*.

The negative SP recorded from scala media in the 5 to 7 day animals had a time course characterized by a rapid onset at the beginning of the tone burst, followed by a slower increase to a maximum value during the time the tone was sounding. It has been suggested originally by von Békésy (1951 c, 1952 a) that the electric energy liberated at the activation of the hair cells is derived from the potential difference between the endolymph and the interior of the hair cells. The dual time course of the early SP could suggest that two different processes are contributing to produce it, *i.e.* one rapid component directly dependent on the stimulation of the hair cells and another caused by a successive slow depletion of EP at a rate, which exceeds the one at which the latter is generated by stria vascularis.

The reversal of SP during the progress of the development of the organ of Corti is consistent with the suggestion of Davis *et al.* (1958 a and b) concerning the origin of SP— and SP+, since several histological investigations (Held 1909, Cajal 1919, Wada 1923, Lorente de Nó 1926, Tello 1931) have shown that the development of the internal hair cells precedes that of the external ones (*cf.* Section VI). Accordingly the early negative SP would be generated by the internal hair cells. Later appearing SP+ from the external hair cells would result in a decrease of the originally negative

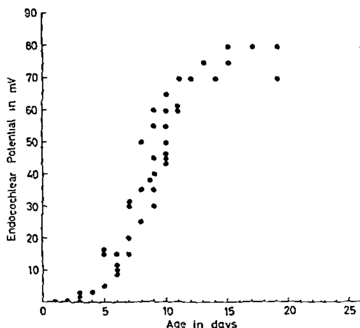



Fig 19 Development of endocochlear potential of the basal turn

cell function of certain poikilothermic vertebrates (Schmidt & Fernandez 1962) It has been shown that CM, and the action potential of the auditory nerve (Tasaki & Fernandez 1952), as well as SP (Davis Eldredge & Gannon 1958) may be augmented by the application of current between electrodes in scala vestibuli and scala tympani in a direction that increases the external polarization of the cochlear hair cells Reversal of the current flow reduces these potentials It has therefore been suggested (Davis 1956, 1957 1960) that EP increases the sensitivity of the cochlear hair cells by adding an external positive polarization to the negative intracellular one, thus increasing the total potential difference over the hair cell membrane

## *Results*

When a glass microelectrode was advanced through the exposed spiral ligament of the basal turn in a rabbit 1 to 3 days old one or several negative resting potentials of about 20 mV were encountered, when the electrode was estimated to be located at the level of stria vascularis As the electrode was advanced, the potential level usually returned very close to zero when the endocochlear space was entered.

In some animals 3 to 4 days old a positive potential of about 3 mV could recorded in scala media In 5-day animals the positive potential of the endocochlear space was found to be 5 to 15 mV With advancing age EP increased as appears Fig 19 A very rapid increase was observed between the age of 5 to 10 days and at age of about 15 days EP attained a level of about 80 mV During the su period no further increase was observed

'ng Su

# III. Endocochlear Potential

The positive DC potential of the endocochlear space, usually referred to as the endocochlear potential (EP), was originally demonstrated by von Békésy (1952 a) He also found that the epithelial structures, forming the walls of the cochlear duct including the hair cells and the cells of stria vascularis, had negative intracellular resting potentials These observations were later confirmed by other investigators (Tasaki *et al* 1954, Gisselsson 1955) The positive DC potential usually amounts to about 80 mV in the basal turn of the guinea pig, somewhat lower values being recorded further apically The positive potential of the vestibular part is of smaller magnitude having a value of about 5 mV (Tasaki 1957, Smith *et al* 1958, Misrahy *et al* 1958, Eldredge *et al* 1961, Schmidt 1963)

A reduction of EP is observed when the respiration of the experimental animal is prevented (Bekesy 1952 a, Davis, Tasaki & Eldredge 1955, Gisselsson 1955, Misrahy *et al* 1958, Rice & Shinabarger 1961) or when the cochlear artery is obstructed (Konishi *et al* 1961) Under these conditions EP may even become negative These observations suggest that EP is dependent on oxidative intermediary metabolism It has, on the other hand, been found that the reduction of EP produced by anoxia may for a considerable time be prevented by perfusion of nitrogen saturated Ringer solution through scala vestibuli (Honrubia *et al* 1962, Butler *et al* 1962) For this reason, it has been suggested that the effect of anoxia on EP may be due to accumulation of ' toxic metabolites '

The rich vascular supply and the histological structure of stria vascularis indicate a site of high metabolic and secretory activity, and it has accordingly been suggested that this epithelium might be the generator of EP (Smith 1957) Several experimental observations confirming this proposition have been presented Thus a more pronounced reduction of EP than of CM and SP was observed by Davis *et al* (1958 b), when stria vascularis had been damaged by surgical obstruction of the cochlear vein Damage to the hair cells produced by the ototoxic action of streptomycin did not affect EP Tasaki and Spyropoulus (1959) recorded a normal EP in waltzing guinea pigs with complete degeneration of the organ of Corti By exploring the inner surface of scala media in normal guinea pigs, after having removed the endolymph, the latter investigators also found that stria vascularis was positive relative to the other structures of the cochlear partition

Bekesy (1952 a) suggested that EP and the intracellular resting potentials of the hair cells could be the sources of the various potentials recorded when the hair cells are activated EP is, however, apparently not essential for hair cell function in the vestibular part of the membranous labyrinth, since, as already mentioned above, the external polarization is very low in this area The same applies to the cochlear hair

of $EP_I$ obtained in the same animal (Fig 20 and 21) $EP_{II}$ started to appear at the same time as $EP_I$ and subsequently increased with an approximately linear relation to $EP_I$ The same seems to apply for $EP_{III}$ but the variation of the values obtained makes this inference less conclusive

The effect of anoxia on EP was studied in 10 animals Anoxia was produced by intraperitoneal administration of a neuromuscular blocking agent (Flaxedil May & Baker Ltd), in a dosage sufficient to produce apnea The rate of decrease of EP could not be determined, because the speed with which the drug acted varied when administered by this route In all animals including the very young ones in which EP was low the potential in the endocochlear space decreased to a level considerably below zero

## *Discussion*

The significance of the small positive potential of the endocochlear space occasionally observed in 3 to 4 day animals is uncertain since potentials of this magnitude might possibly represent changes of the junction potential at the electrode tip In view of the low grid current ($10^{-13}$ A) of the input stage of the amplifier, it is unlikely that potentials recorded along the electrode track would have been due to impedance changes at the electrode tip At an age of 5 days an obvious increase of the positive potentials suggests the appearance of EP, which subsequently increases to attain a level of about 80 mV from the age of 15 days This level is comparable with the one recorded in adult mammals (*cf* Schmidt & Fernandez 1962) The development of EP may therefore be considered to be completed at this age

Since stria vascularis is considered to be the generator of EP it is interesting to note that the cells of this epithelium were found to have a negative resting potential, even before the appearance of EP The increase in magnitude of the negative resting potentials in connection with the increase of EP suggests an increase of the capacity of stria vascularis to generate a potential difference between the epithelium and the endolymph The maintenance of EP requires in addition that the structures forming the borders of the endocochlear space develop a resistance sufficiently high to maintain the imposed positive potential Whether this occurs before or after the appearance of EP cannot be established on the basis of the present observations

Schmidt & Fernandez (1963) have studied the development of EP in the opossum, the rat and the mouse In the two latter species only a few preliminary experiments were performed In the opossum the relation of the development of EP to the morphological differentiation of the inner ear seems to be [illegible]
found in the rabbit Schmidt & Fernandez compared their observat[illegible]
in the opossum with the data of McCrady *et al* (1937, 1940) on the [illegible]
of CM in the same animal The correlation was found to be good [illegible]
early period of the development, during which EP was observed [illegible]
than CM It was considered that this discrepancy might be due [illegible]

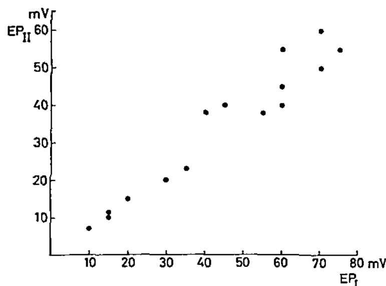



Fig 20 Development of endocochlear potential of the middle turn ($EP_{II}$) in relation to endocochlear potential of the basal turn ($EP_{I}$)

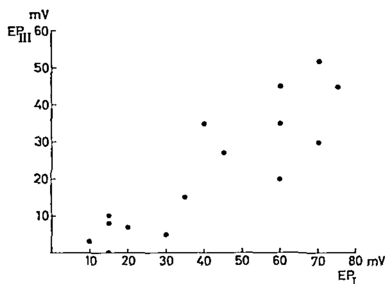



Fig 21 Development of endocochlear potential of the apical turn ($EP_{III}$) in relation to endocochlear potential of the basal turn ($EP_{I}$)

Definite values cannot be given on the development of the negative resting potentials of stria vascularis, since the magnitude of these potentials varied somewhat with the size of the electrode tip The observations did, however, suggest that these potentials increase in magnitude during development to a value of at least 50 mV

In some of the experiments EP was also measured in the middle turn ($EP_{II}$) and in the apical turn ($EP_{III}$) As EP of the basal turn ($EP_{I}$) has been shown to increase with advancing age, and as the values of $EP_{I}$ within each age group probably to some extent reflects the difference in maturation that can be expected to take place within the course of one day $EP_{II}$ and $EP_{III}$ have been plotted against the value

of $EP_I$ obtained in the same animal (Fig 20 and 21) $EP_{II}$ started to appear at the same time as $EP_I$ and subsequently increased with an approximately linear relation to $EP_I$ The same seems to apply for $EP_{III}$ but the variation of the values obtained makes this inference less conclusive

The effect of anoxia on EP was studied in 10 animals Anoxia was produced by intraperitoneal administration of a neuromuscular blocking agent (Flaxedil May & Baker Ltd), in a dosage sufficient to produce apnea The rate of decrease of EP could not be determined, because the speed with which the drug acted varied when administered by this route In all animals including the very young ones in which EP was low the potential in the endocochlear space decreased to a level considerably below zero

## Discussion

The significance of the small positive potential of the endocochlear space, occasionally observed in 3 to 4 day animals is uncertain since potentials of this magnitude might possibly represent changes of the junction potential at the electrode tip In view of the low grid current ($10^{-1?}$ A) of the input stage of the amplifier, it is unlikely that potentials recorded along the electrode track would have been due to impedance changes at the electrode tip At an age of 5 days an obvious increase of the positive potentials suggests the appearance of EP, which subsequently increases to attain a level of about 80 mV from the age of 15 days This level is comparable with the one recorded in adult mammals (*cf* Schmidt & Fernandez 1962) The development of EP may therefore be considered to be completed at this age

Since stria vascularis is considered to be the generator of EP, it is interesting to note that the cells of this epithelium were found to have a negative resting potential, even before the appearance of EP The increase in magnitude of the negative resting potentials, in connection with the increase of EP suggests an increase of the capacity of stria vascularis to generate a potential difference between the epithelium and the endolymph The maintenance of EP requires in addition that the structures forming the borders of the endocochlear space develop a resistance sufficiently high to maintain the imposed positive potential Whether this occurs before or after the appearance of EP cannot be established on the basis of the present observations

Schmidt & Fernandez (1963) have studied the development of EP in the opossum, the rat and the mouse In the two latter species only a few preliminary experiments were performed In the opossum the relation of the development of EP to the morphological differentiation of the inner ear seems to be similar to that found in the rabbit Schmidt & Fernandez compared their observations on EP in the opossum with the data of McCrady *et al* (1937, 1940) on the development of CM in the same animal The correlation was found to be good except during the early period of the development, during which EP was observed to appear earlier than CM It was considered that this discrepancy might be due to the fact that up

to the age at which McCrady *et al* had found CM to appear, the external auditory meatus was observed to be closed, thus preventing satisfactory stimulation of the inner ear This argument is in agreement with the view previously expressed in the present paper (p 6 and p 26) The limited number of measurements presented by Schmidt & Fernandez on the development of EP in the mouse and the rat suggested that this potential is established during the first two weeks after birth There is apparently at least a rough similarity in this respect between these two species and the rabbit

In the present investigation the time course of development of EP was observed to coincide with that of the receptor potentials The possibility that the appearance of EP would initiate receptor activity in the organ of Corti seems unlikely in view of the observations that other types of hair cells, for instance in the vestibular part of the labyrinth, operate without or with very low external polarization On the other hand, it seems likely that EP participates to produce the gradual increase of the sensitivity of the cochlear hair cells observed during development

# IV. Electric Response of Second Order Auditory Neurons

In the previous sections the development of the receptor potentials and the endocochlear potential has been described The investigations reported below were undertaken in order to determine if the hair cells at the time of appearance of these phenomena are capable of generating nerve impulses, and to study some aspects of the impulse generation and propagation during the development

## *Results*

Attempts were made to record the action potential of the auditory nerve from the round window in response to click stimulation Differential recording from the basal turn according to the method described by Tasaki, Davis & Legouix (1952) was also tried However, under the specific recording conditions prevailing in the undeveloped ear it was found that the success, with which action potentials could be recorded with these methods during the early period of receptor development, was too variable to permit a systematic investigation The possibility of recording from the surgically exposed auditory nerve was excluded, since anatomical considerations indicated difficulties in preventing damage to the vascular supply of the inner ear

For this reason it was found preferable to employ stereotactic recording technique A suitable method was devised, to meet the demands imposed by the gradual increase in size of the skulls of the experimental animals during the development The details *of the technique have been described under Methods*

The intention was to place the electrode tip in the auditory nerve However, the histological controls, which were not available for study until after completion of the experimental series, revealed that the electrode tracks were situated somewhat medially to the internal auditory meatus The electrode tips were actually found to impinge on *the region where the fibres of the trapezoid body leave the cochlear nuclear complex* Observations of systematic changes of the configuration of the potentials recorded along the electrode track allowed reproduceable localization of the electrode tip

An example of the type of potentials recorded along the electrode track in response to the standard 3 kc tone burst at 125 db is given in Fig 22, obtained from an experiment on a 10 day rabbit In general the following types of slow wave potentials were observed in these experiments Along the first half of the track, as the electrode passed through the cerebellar hemisphere no responses were observed As the electrode approached the posterior aspect of the dorsal cochlear nucleus a response appeared, and subsequently increased in magnitude when the electrode entered the nucleus This response was characterized by an initial positivity followed by a prolonged nega-

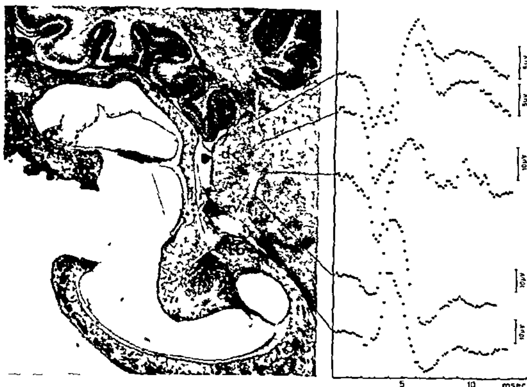



Fig 22 Averaged responses observed in 10 day rabbit at indicated positions along electrode track Stimulation standard 3 kc tone burst at 125 db CM appeared 0 5 msec after beginning of records Upward deflection indicates negativity at recording electrode Calibration bars refer to average magnitudes of individual responses Abbreviations dc, dorsal cochlear nucleus, av, anteroventral cochlear nucleus, tb, trapezoid body, V, descending root of trigeminal nerve *Footplate of the stapes removed at fixation*

tivity From the age of about 10 days additional superimposed deflections were observed, like those present in the upper record of Fig 22 With increasing age the complexity of the wave form increased When the electrode was further advanced the response gradually changed into the shape of a triphasic potential of the type shown in the two lowest records of Fig 22 As mentioned, this response configuration was observed when the electrode tip was situated in the area where efferent fibres from the second order auditory neurons collect in forming the trapezoid body This type of response could be reproduced without changes of the configuration during the entire developmental course, and was therefore selected for study The following account will be restricted to this type of response unless otherwise stated

The amplitude of the potential did not show any consistent relation to the age of the experimental animal, but rather depended upon the proximity of the electrode to *the center of the target area* Since the amplitude *was frequently only of the order of* about 10 $\mu$V, it was found convenient to use the computer of average transients (see Methods) in order to obtain records suitable for latency measurements

The first experiments were performed on 5 day animals, since at this age the receptor potentials had been found to appear The animals were tested with tone bursts

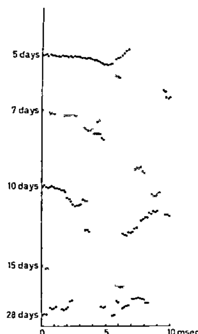



Fig 23 Averaged responses recorded at the trapezoid body in animals of indicated ages Stimulation standard 3 kc tone burst at 125 db CM appeared 0 5 msec after beginning of records Upward deflection indicates negativity at recording electrode

of different frequencies between 0 5 and 10 kc presented at the maximally available sound intensity The 3 kc tone burst described under Methods was found to produce an optimally large and synchronized action potential For this reason it was chosen as a standard stimulus for this part of the investigation It was ascertained that the repetition rate of 5/sec at which the tone bursts were presented in order to feed the responses into the computer, did not affect the latency or the amplitude of the response

A triphasic potential of the type described above was recorded in five out of seven 5 day animals tested with the 3 kc tone burst One 3 day animal and four 4 day animals did not show activity at any level along the electrode track A response was recorded in all animals older than 5 days

Records of potentials obtained in animals representing five different stages of development are shown in Fig 23 There is a delay of 0 5 msec between the beginning of the record and the appearance of CM at the round window This delay represents the time needed for triggering of the gate and for the acoustic transmission to the inner ear Latencies were measured from the appearance of CM to the peak of the initial positivity and to the peak of the following negativity Fig 24 shows the values obtained in the entire group of animals studied in this respect From the age of 5 days the latency of the positive peak decreased from 3-4 5 msec and attained a value of about 1 5 msec at an age of 15 days During this period the latency of the negative peak

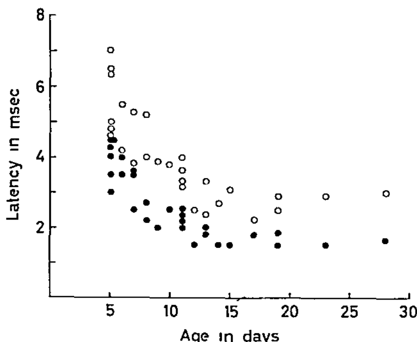



Fig 24 Development of latencies of responses recorded at the trapezoid body Filled circles latency of initial positive peak. Open circles latency of following negative peak Stimulation standard 3 kc tone burst at 125 db

was found to decrease from 4 5-7 msec, reaching a final value of 2 5 3 msec at the end of it

Animals were also tested for responsiveness to a click stimulus of the type specified under Methods The intention was to present a stimulus with a wide frequency content, which would activate a larger population of hair cells than the tone burst However, in 5-day animals, which were found to respond to the 3 kc tone burst, the click failed to evoke any response A response to the click could not be recorded until the age of 8 days At that age the potential had only half the amplitude of the one recorded in response to the tone burst From the age of 10 days the amplitude of the potential evoked in response to the click was of the same order of magnitude as the one elicited by the tone burst The response evoked by the click had the same latency as the one recorded in response to the tone burst

In order to find out whether the difference in responsiveness to the two types of stimuli was due to their different durations the following experiments were performed The rise and fall time of the 3 kc tone burst was adjusted so that full amplitude was retained throughout the time course of the signal The duration was reduced so that the stimulus only contained 1½ cycles This was the shortest duration available and actually made the signal more like a click than a tone burst The amplitude of the potential was then studied in relation to increasing duration of the signal at half cycle intervals As the magnitude of the initial positive and the negative components of the potential varied proportionately, it was found convenient to measure the amplitude of the potential between the peaks of these two components The experiments were per-

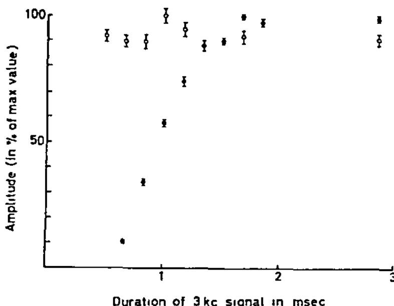



Fig 25 Effect of increasing duration of 3 kc signal (see text) on amplitude of the response recorded at the trapezoid body in a 5-day rabbit (filled circles) and in a 15-day rabbit (open circles) Bars indicate ± one standard error of the mean calculated from ten consecutive records

formed on animals in which a large action potential could be recorded directly on the cathode ray oscilloscope At each stimulus duration a series of ten responses were photographed for measurements Fig 25 illustrates the results obtained from two animals, 5 and 15 days old, respectively In the 5 day animal a response could not be detected until the signal had a duration corresponding to two complete cycles With further prolongation of the signal duration the amplitude of the response increased up to a maximum which was attained when the signal duration exceeded a value corresponding to five cycles A similar result was obtained in another 5 day animal One 7 day animal was found to be slightly more responsive to the short signals than the 5-day animals By the end of the developmental period the response retained practically the same amplitude when the duration of the signal was changed (*cf* 15 day animal in Fig 25) The implications of these observations will be further discussed below

## *Discussion*

The electric responses evoked in the cochlear nuclear complex in response to clicks have been studied in cats by several investigators (Kemp Coppee & Robinson 1937 Ades & Brookhart 1950 Jungert 1958 Rose Galambos & Hughes 1959 Moushegian Rupert & Galambos 1962 Rupert, Moushegian & Galambos 1963 Altman Radionova & Ratnikova 1964) Corresponding to the complex microanatomy of

the cochlear nuclei, the configuration of the response changes considerably in different parts of this region The sources of the different components of the response have not been identified However it is generally considered that the response arises as the result of the activation of the second order auditory neurons In spite of systematic changes of the configuration of the response described by Galambos and his associates (1962, 1963) and by Altman *et al* (1964), it is difficult to compare the records published by the above investigators However, the latency of the first component of the responses recorded in the cochlear nuclei and their efferent pathways is generally stated to be about 1 5 1 7 msec In the present investigation values of this order of magnitude were observed from the age of about 15 days It may therefore be concluded that from the age of 15 days the response has attained mature characteristics as far as latency is concerned

The reduction of the latency of the second order neuronal response observed during development, indicates that the efficiency of impulse transmission from the receptor level gradually increases up to an age of 15 days To what extent this reduction is due to decreased delay of the impulse generation in the organ of Corti, increased conduction velocity of the auditory nerve and/or decrease of the synaptic delay between the first and second order neurons cannot be evaluated on the basis of the present observations However, a few experiments, in which the action potential of the auditory nerve was recorded, indicated that in developing animals the latency is prolonged also at the level of the first auditory neuron This is in agreement with the observations of Rose, Adrian & Santibanez (1957) and Khavina & Maruseva (1963), who during the postnatal development of kittens observed a reduction of the latency of the neural component of the round window response to clicks Thus, Rose *et al* (1957) reported that the peak latency of the first neural component of the response, which appeared 11 days postnatally, decreased from a value of 2 7 msec (corrected for acoustic delay) to attain an adult value of 1 5 msec at an age of 21 days In a similar investigation in young mice Alford & Ruben (1963) observed a cochlear microphonic potential at an age of 9 to 13 days and a neural response, on the average, 1 5 days later The latency of this neural response subsequently decreased from 1 3 msec to 1 0 msec Similar latencies were observed in a later investigation in the same species (Mikaelian & Ruben 1964) This latency reduction is very moderate, which may indicate that the responses were first detected at a comparatively late phase of the initial development

As reported above clicks, in contrast to tone bursts, did not evoke a response in the second order auditory neurons during the first three days following the inception of the functional development of the inner ear During this period the duration of the stimulating signal obviously has to exceed a certain minimum value in order to produce a neural response at the level where the neural activity was studied in the present investigation This finding emphasizes the importance of selecting appropriate stimulation conditions in order to study the early development of the inner ear function The possible error introduced by the concomittant development of the external and middle ear has been pointed out earlier in this paper (see pp 6 and 26) It seems likely that in previous investigations (see pp 5-6), in which stimulation with

clicks were used to evoke a response in the auditory nerve or in the cortex the early stages of the functional development of the inner ear have escaped recognition

In rabbits 5 to 7 days old an *increase of the duration of the stimulating signal* beyond the minimum duration required for the production of a response in the second order auditory neuron was shown to result in a gradual increase in the amplitude of the response up to a maximum The most immediate explanation of this effect would be that *increasing duration of the stimulating signal results in activation of a larger* number of neurons and/or in increasing synchronization of the initial neuronal activation Since the phenomenon was observed at the level of the second order neurons it cannot be decided whether the underlying mechanism is situated in the organ of *Corti or in the cochlear nuclei When comparing these results with those obtained* by the end of the second postnatal week it appears that the efficiency of impulse transmission gradually increases during the development to attain essentially adult character during the latter period

*The age of appearance of evoked activity in the second order auditory neurons coin*cides with that at which the receptor potentials and the endocochlear potential were observed to appear The auditory nerve fibres are obviously ready to serve when the receptor elements start to acquire functional capacity This is in agreement with the *opinion expressed by Marty (1962) and Marty & Thomas (1963) who from obser*vations of responses in the auditory cortical area concluded that the development of the central auditory pathways precedes that of the receptor function Thus electric stimulation of the auditory nerve in kittens was reported to evoke a response at birth *whereas stimulation with clicks did not produce a cortical response* until an age of 8 days In both species and particularly in rabbits the latency and the configuration of the response evoked by natural stimulation indicated that at the time of its appearance the central auditory pathways were in an advanced stage of development

# V. Appearance of the Acoustic Reflex of the Middle Ear Muscles

The acoustic reflex of the middle ear muscles is known to be sensitive to general anesthesia This has also been observed in the rabbit (Wersall 1958, Moller 1964, 1965) In view of the urethane anesthesia employed in the present investigation it was therefore surprising to find that during the surgical preparation of the middle ear contractions of the tensor tympani muscle could frequently be observed from the age of 5 days The contractions occurred in connection with surgical manipulation of the stapes or when trimming away bone over the spiral ligament The stapedius muscle responded less readily but was on the other hand, difficult to observe, as it is situated in a deep recess It was undoubtedly the acoustic quality of this crude type of stimulation which produced the reflex response, since receptors representing other modalities are not present in the deep layer of the otic capsule The threshold was high, since tone bursts presented at maximum available intensity frequently failed to elicit a reflex response

The observation supports the conclusion (see p 45) that central auditory pathways are ready to serve when the receptor function of the inner ear appears

# IV. Morphological Development

## *Comment on the Development of the Middle Ear*

The necessity of removing the middle ear in order to obtain comparable stimulation conditions during the entire developmental period has been pointed out earlier in this paper. A short description of the development of the middle ear in the rabbit will therefore be given. The ossification of the otic capsule, the bulla and the auditory ossicles in the rabbit has been described by Hoyte (1961).

During the surgical preparation of the middle ear the footplate of the stapes was observed to be equally mobile during the entire developmental period studied.

Up to the age of 5 days the middle ear of the young rabbit (Fig. 26 a) is occupied by mesenchymal connective tissue into which a small extension of the pharyngeal pouch projects, at this stage still filled with serous fluid. The ossification of the ossicles has started but certain parts such as the manubrium of the malleus are still cartilaginous and flaccid. The external auditory meatus lacks osseous and cartilaginous support and on inspection it appears as a closed slit. During the following 5 days the pharyngeal pouch rapidly expands at the expense of the mesenchymal tissue. At 7 to 8 days the serous fluid of the pouch has largely been replaced by air. However, mesenchymal tissue is still found around the stapes, in the epitympanic recess and in the round window niche. At 10 days the middle ear is almost completely pneumatized (Fig. 26 b) and the osseous support of the auditory meatus is being formed.

## *Organ of Corti*

The morphological development of the ear has been extensively studied (*cf.* Kolmer 1927, and Bast & Anson 1949). The development of the organ of Corti in the rabbit from the stage attained at the end of the gestational period to the fully developed sense organ has been meticulously described by Retzius (1884) and Held (1909). The present histological observations agree in all essential respects with the descriptions and illustrations presented by these authors. For this reason the following account will be restricted to certain features of the morphological development, which appear to be pertinent to the functional development.

As generally recognized, the development of the organ of Corti in all mammals studied (with the single exception of the opossum, Larsell, McCrady & Zimmermann 1935, Larsell, McCrady & Larsell, 1944) proceeds from the base towards the apex. In the rabbit this sequence of development is clearly brought out in the illustrations of Retzius (1884), showing the organ of Corti in the basal, middle and apical turn at the term of the gestational period as well as in a new born animal and in animals representing postnatal ages of 2, 7, 10 and 14 days. The development in the basal




Fig 26 Sections through the middle ear of 5 day (a) and 10 day (b) rabbits Bar 1 mm Magnification 8 ×

turn precedes that of the middle turn by about one day and that of the apical turn by about two days

The progress of the development of the organ of Corti with increasing age is illustrated in Fig 27, which shows the organ of Corti of the upper part of the basal turn in a new born animal and at postnatal ages of 4 5 7 11 and 18 days The appearance differs only to a minor extent from that in the lower part of the basal turn

*New born animal* The organ of Corti shows obvious immature features The future internal spiral sulcus is occupied by a tall pseudostratified epithelium The internal and external hair cells are easily recognized The cuticular plates of the external hair cells stand parallel with the basilar membrane while that of the internal hair cells forms an obtuse angle with the external part of the reticular membrane The external hair cells have a more rounded appearance than in the adult animal The internal and external pillar cells are wedge shaped and situated close together No signs of the future tunnel of Corti are yet visible Deiter s and Hensen s cells have small dimensions Nuel s spaces are not present The tectorial membrane is thinner than in the subsequent stages At the internal margin of the pseudostratified epithelium the tectorial membrane is detached From the external margin of the tectorial membrane thin strands project towards the cuticular plates of the supporting cells as described by Held (1909) The thick tympanic covering layer of the basilar membrane consists of densely packed cells Vas spirale is conspicuous In pars pectinata of the basilar membrane elongated nuclei are seen External to Hensen s cells the basilar membrane is covered with a low cuboidal epithelium

*4 day animal* The most striking feature is the beginning of regressional changes at the internal margin of the pseudostratified epithelium As this process proceeds the internal spiral sulcus is successively covered by a low cuboidal epithelium The bases of the pillars have started to separate radially The fibrillar structure of the pillar cells is visible as denser areas within the cytoplasm At the place of the future tunnel of Corti and the first space of Nuel the cytoplasm is clearer but well defined intercellular spaces cannot yet be seen The tectorial membrane has attained a shape closely similar to the final one

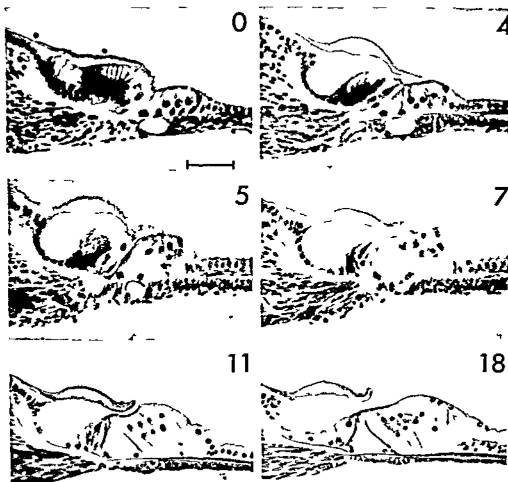



Fig 27 Development of the organ of Corti. Upper basal turn. Numbers indicate age in days. Bar 50 $\mu$. Magnification 240 ×

*5 day animal* The regression of the pseudostratified epithelium has progressed leaving a mass of decomposing cell elements. The bases of the pillar cells have separated further. The base of the internal pillar has moved closer to the habenula perforata and that of the external pillar has been displaced outwards. The tunnel of Corti is clearly visible between them. The first space of Nuel has been formed. The organ of Corti has increased in height mainly as a result of elongation of the external pillar and Deiter's cells.

*7 day animal* The organ of Corti is approaching adult appearance. The pseudostratified epithelium has almost completely disappeared and only a few pyknotic nuclei still remain close to the internal hair cell. As a result of further elongation of the external supporting elements the reticular membrane slopes inwards and the cuticular plates of the internal and external hair cells are at this stage in the same plane




Fig 26 Sections through the middle ear of 5 day (a) and 10 day (b) rabbits Bar 1 mm Magnification 8 ×

turn precedes that of the middle turn by about one day and that of the apical turn by about two days

The progress of the development of the organ of Corti with increasing age is illustrated in Fig 27, which shows the organ of Corti of the upper part of the basal turn in a new-born animal and at postnatal ages of 4, 5, 7, 11 and 18 days The appearance differs only to a minor extent from that in the lower part of the basal turn

*New born animal* The organ of Corti shows obvious immature features The future *internal spiral sulcus* is occupied by *a tall pseudostratified epithelium* The *internal* and external hair cells are easily recognized The cuticular plates of the external hair cells stand parallel with the basilar membrane, while that of the internal hair cells forms an obtuse angle with the external part of the reticular membrane The external hair cells have a more rounded appearance than in the adult animal The internal *and external pillar cells are wedge shaped and situated close together No signs of the* future tunnel of Corti are yet visible Deiter's and Hensen s cells have small dimensions Nuel s spaces are not present The tectorial membrane is thinner than in the subsequent stages At the internal margin of the pseudostratified epithelium the tectorial membrane is detached From the external margin of the tectorial membrane *thin strands project towards the cuticular plates of the supporting cells* as described by Held (1909) The thick tympanic covering layer of the basilar membrane consists of densely packed cells Vas spirale is conspicuous In pars pectinata of the basilar membrane elongated nuclei are seen External to Hensen's cells the basilar membrane is covered with a low cuboidal epithelium

*4-day animal* *The most striking feature is the beginning of regressional changes at* the internal margin of the pseudostratified epithelium As this process proceeds the internal spiral sulcus is successively covered by a low cuboidal epithelium The bases of the pillars have started to separate radially The fibrillar structure of the pillar cells is visible as denser areas within the cytoplasm At the place of the future tunnel of Corti and the first space of Nuel the cytoplasm is clearer but well defined *intercellular* spaces cannot yet be seen The tectorial membrane has attained a shape closely similar to the final one

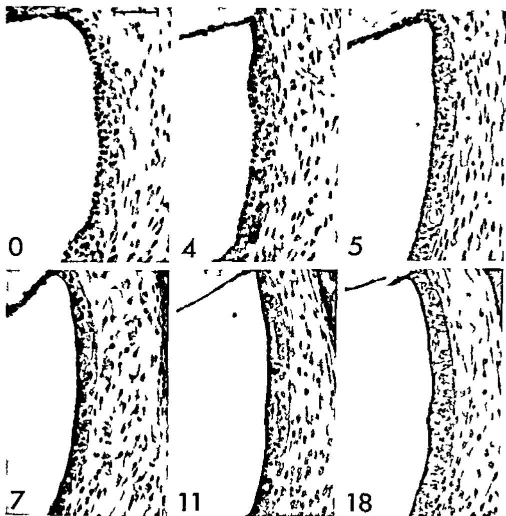



Fig 28 Development of stria vascularis Upper basal turn Numbers indicate age in days Bar 50 $\mu$ Magnification 240 ×

the spiral ligament by flattened cells of mesenchymal origin

The postnatal development of stria vascularis in the rabbit is illustrated in Fig 28 with sections from the upper part of the basal turn

*New born animal* The development attained at birth represents a fairly advanced stage of the process described above Below the dark superficial cells an aggregation of lighter cells with large pale nuclei are situated A distinct basement membrane can not be seen between the two groups of cells The border against the connective tissue of the spiral ligament also appears indistinct

*4 day animal* The deeper layer of stria vascularis can be more clearly distinguished from the spiral ligament than in the previous stage

*5 to 18 day animals* The superficial cells gradually acquire a darker cytoplasm and

The pillars have not yet attained their final delicate shape The tunnel of Corti and the first space of Nuel have expanded In addition Nuel s spaces are visible around the external two rows of hair cells The external hair cells are more slender than in the previous stages Vas spirale is considerable smaller than before

*11 day animal* The structure of the organ of Corti can hardly be distinguished from that of the adult animal. The internal spiral sulcus and the intercellular fluid spaces of the organ of Corti are completely developed. However the cells of the internal spiral sulcus and Hensen s cells have not yet attained their final size Moreover the tympanic covering layer is further reduced in thickness but is still thicker than in the adult

*18 day animal* The organ of Corti has attained adult structure over the entire cochlear duct. The cells of the internal spiral sulcus and Hensen s cells have increased in size The cells of the previously cuboidal epithelium external to the organ of Corti are now more polygonal and covered by Hensen s cells and Claudius cells thus revealing their identity with Boettcher s cells

## *Stria Vascularis*

The development of stria vascularis has previously been studied in several species but not in the rabbit. The literature has been reviewed by Shambough (1907) von Fieandt & Saxen (1937 a) and Weibel (1957) Electron microscopic investigations on the adult structure of stria vascularis (Engstrom Sjostrand & Spoendlin 1955 Smith 1957) have confirmed and extended earlier light microscopic observations

As mentioned previously (p 34) stria vascularis is considered to be the generator of the endocochlear potential It has further been suggested that this highly specialized epithelium participates in the regulation of the chemical composition of the endolymph and in the transport of oxygen to the endocochlear space (*cf* Rauch *et al* 1964)

From the histological investigations referred to above it appears that stria vascularis consists of superficial cells with dark cytoplasm and deeper cells of lighter appearance The superficial cells send cytoplasmic extensions into the deep layer where they interdigitate with the second type of cells The cells of stria vascularis are in close contact with intraepithelially situated capillaries

The superficial cells derive from the primordial epithelium in the lateral wall of the cochlear duct Divergent opinions have been expressed concerning the origin of the cells situated in the deeper layers of stria vascularis One group of investigators (von Fieandt & Saxen 1937 and others) consider stria vascularis to be of epithelial origin. Another group (Shambough 1907 Weibel 1957 and others) regard some of the deeper epithelial cells to be of mesodermal origin.

According to Shambough (1907) and Weibel (1957) the primordial epithelium is delimited by a well defined basement membrane. Later mesenchymal cells and capillaries aggregate below the basement membrane which is subsequently disrupted whereupon the two types of cells and the capillaries begin to develop their complicated structural relations By the end of this process stria vascularis is delimited from

ligament from the region immediately below the spiral prominence From the investigation of Shambough (1909) these cells are often known in the anglo saxon literature as the glands of Shambough A systematic study of the development of these structures in the mouse has more recently been presented by Weibel (1957)

The function of these cells is not known The structure and the histo-chemically demonstrated presence of succinic dehydrogenase (Vosteen 1961, 1964) suggest a site of high metabolic activity Their possible role for the regulation of the composition of the endolymph has been discussed by Rauch (1964)

These epithelial elements are generally described to originate from th primordial epithelium, from which the extensions into the spiral ligament gradually develop

The development of the epithelium at the external spiral sulcus of the basal turn is illustrated in Fig 29 In the new born animal circumscribed groups of cells are seen to invade the spiral ligament in the region below the spiral prominence In the following stages these cells spread further into the spiral ligament insinuating themselves between the connective tissue cells As the development progresses it becomes more difficult to distinguish the epithelial extensions from the connective tissue cells In the two latest stages represented in Fig 29 it is difficult to state how far the epithelial cells actually extend A darker area, possibly indicating the extension of the epithelial cells, reaches up to the level of the lower third of stria vascularis (*cf* Fig 28) Claudius' cells develop from the low cuboidal cells situated above the attachement of the basilar membrane to the spiral ligament These cells gradually increase in size and extend, in the final stages, out over the basilar membrane covering the small polygonal cells on the basilar membrane, the so-called Boettcher's cells

## *Reissner's Membrane*

Reissner s membrane constitutes a barrier between the endolymphatic and perilymphatic spaces This membrane contributes to the maintenance of the endocochlear potential and the ionic composition of the endolymph (*cf* Rauch *et al* 1964)

Reissner's membrane develops from the epithelium of the vestibular border of the primordial cochlear duct On the vestibular aspect of this epithelium cells from the mesenchymal periotic reticulum aggregate (*cf* Bast & Anson 1949)

The development of Reissner s membrane is seen in Fig 28 During the development the thickness of the membrane is gradually reduced

# General Discussion

The structure of the developing inner ear, as it appears with light microscopical technique, allows only limited conclusions concerning the functional ability of the various constituents. It is therefore not possible to point at any particular feature of the structural development as being responsible for the initiation of the cochlear function, particularly in view of the probability that the development of several equally important parts of the sensory organ may coincide, as does for instance the development of the middle and the inner ear.

However, in spite of these limitations, certain features of the developing organ of Corti seem to be of rather obvious importance for its functional ability. Thus the presence of the tall pseudostratified epithelium in the internal spiral sulcus can reasonably be assumed to impede the transverse rocking movements of the organ of Corti. The importance of these movements for the production of the shearing forces acting as stimulus for the hair cells has been pointed out by von Bekesy (1953 a and b). The residues of the pseudostratified epithelium, present at the initial stages at which cochlear function was observed to be present, may partly be responsible for the low efficiency of intracochlear sound transmission to the hair cells at this stage (p. 26). The same effect may also be attributed to the immaturity of the supporting structures of the organ of Corti.

The tunnel of Corti and the spaces of Nuel appear in connection with the initiation of cochlear function. The fluid confined within these intercellular spaces has been named cortilymph by Engstrom (1960 a and b). As appears from the review by Rauch (1964) our knowledge concerning the formation and resorption of this fluid as well as its significance for the function of the organ of Corti still presents many controversial aspects. It is, however, generally agreed that this fluid, in contrast to the endolymph, has a low concentration of potassium since transmission in the terminal nerve fibres crossing these spaces would otherwise be impossible (Tasaki, Davis & Eldredge 1954). It is not possible to decide whether the appearance of these spaces is of importance for the inception of cochlear function or if they appear as a secondary consequence of the development of the supporting structures of the organ of Corti.

The histological technique employed in the present investigation does not permit any statement on the detailed structural differentiation of the hair cells. However, several previous investigations have shown that the differentiation of the internal hair cells precedes that of the external hair cells (Held 1909, Cajal 1919, Wada 1923, Lorente de No 1926, Tello 1931). In rabbit fetuses at term it was demonstrated by Held (1909) that the development of the cuticular plates and the cilia occurs earlier in the internal hair cells than in the external ones. He also stated that the development of the supporting cells proceeds in the same order. Cajal (1919), Lorente de No (1926) and Tello (1931) observed in studies on the development of the auditory nerve fibres

in mice that the internal hair cells receive their innervation before the external hair cells Tello in addition paid particular attention to the development of certain argentophil reticular structures within the hair cells and observed that these structures develop earlier in the internal hair cells than in the external ones On the basis of measurements of the dimensional increase of the hair cells during the postnatal development of the inner ear in the rat Wada (1923) concluded that the internal hair cells attain their final size earlier than the external hair cells Although the observations referred to above were made mainly during earlier stages of development than that at which the cochlear function was observed to appear in the present investigation it seems likely that the subsequent development at the ultrastructural and biochemical level proceeds in the same sequence

The studies on the development of the auditory nerve fibres referred to above (Cajal 1919 Lorente de No 1926 Tello 1931) show that in mice the primordial epithelium of the organ of Corti is invaded by nerve fibres at a stage at which the hair cells are still not differentiated from the rest of the epithelium In the new born mouse where the development of the organ of Corti is somewhat less advanced than in the rabbit ramifications of nerve fibres were observed around the internal and external hair cells During the following few days a further extension of the spiral nerve fibres took place An electron microscopic study is in progress to investigate at which stage of development synaptic contact between the hair cells and the terminal auditory fibres is established in the rabbit At present the time relation between the appearance of activity in the auditory nerve and the formation of the synapses is not known

Previous investigations in which the morphological development of the organ of Corti has been correlated to results of various types of functional tests (see pp 5 6 Wada 1923 Larsell McCrady & Zimmermann 1935 Larsell McCrady & Larsell 1944 Alford & Ruben 1963 Mikaelian & Ruben 1964) are in general in agreement with the results of the present study However due to the limitations of the stimulation technique employed in these investigations (see pp 6) it has however not been clear whether failure to respond to sound stimulation was due to immaturity of the middle ear and/or the inner ear In view of the results of the present study this ambiguity no longer exists The final formation of the inner spiral sulcus the appearance of the intercellular fluid spaces of the organ of Corti as well as the formation of its supporting elements may consequently be considered to coincide with the appearance of cochlear function Thus Wada (1923) in his studies in the rat observed the organ of Corti to have attained this stage of development when the animals started to exhibit behavioral responses to sound stimulation In the opossum Larsell McCrady & Larsell (1944) arrived at the same result when comparing their histological studies with the appearance of behavioral responses (Larsell Mc Crady & Zimmermann 1936 McCrady Wever & Bray 1937) and the appearance of cochlear microphonic potentials (McCrady Wever & Bray 1937 McCrady Wever & Bray 1940) Alford & Ruben (1963) working with mice did not observe any difference between the structure of the organ of Corti in those animals in which a cochlear microphonic potential was observed and those in which in addition a neural response was found to be present The structural changes occurring in relation to the appearance of the cochlear

microphonic potential were not described. The cochlear microphonic potential could be observed from the age of 9 days (9 to 13 days with a mean of 11.6 days). In a later electrophysiological study on the development of the inner ear function in normal CBA J mice and shaker-1 mice (Mikaelian & Ruben 1964) the cochlear microphonic potential was stated to appear at an age of 8 days in normal mice. According to the description given by Weibel (1957) the organ of Corti has at this age attained a structure comparable to the one which in the present study was observed in 5 day rabbits.

In the opossum the changes taking place in stria vascularis in connection with the development of the endocochlear potential (Schmidt & Fernandez 1963) are similar to those observed in the rabbit.

As generally known (*cf.* Kolmer 1927, Bast & Anson 1949) the development of the inner ear in human fetuses has at the beginning of the sixth month proceeded to the stage at which the internal spiral sulcus and the intercellular spaces of the organ of Corti begin to form. At the end of this month the structure is comparable to the adult one. It would consequently be expected that responsiveness to acoustic stimulation would appear during this month. This has been confirmed with a recently described clinical method for prenatal recording of auditory responses in human fetuses (Johansson, Wedenberg & Westin 1964). The method is based on previous observations showing that prematures with a gestational age of 27 to 28 weeks react to acoustic stimulation by a change of the heart rate. Recently prenatal responses have been observed from the age of 26 weeks (Johansson & Wedenberg 1965).

# Summary

The aim of the present investigation was to study the ontogenetic development of the cochlear function The experiments were performed on young rabbits during the first month after birth Sound stimulation was applied by means of a closed acoustic system sealed over the surgically exposed oval window in order to eliminate the influence related to development of the external and middle ear

I The cochlear microphonic potential (CM) was recorded from the round window in response to tone stimulation between 0 2 and 10 kc CM appeared at an age of 5 days and only occasionally one day earlier In the youngest animals CM was present only in the middle frequency range (about 2 to 5 kc) During the subsequent development the frequency range was extended to lower and higher frequencies The sound intensity required to produce CM with a magnitude of 2 μV r m s gradually decreased and attained a steady level at an age of 15 to 20 days During the entire developmental period there was a simple logarithmic relation between CM and the sound intensity in the lowest intensity range The sound intensity at which CM deviated by 2 db from the linear course decreased and attained a steady level at an age of about 15 days The magnitude of CM at this sound intensity increased during the same period

These observations show that during the development the intensity function of CM is shifted upwards and to the left in the intensity voltage diagram The vertical shift is probably in part due to an increased electric insulation of the cochlea as a result of the gradual ossification of the otic capsule, it presumably also indicates an augmented individual output of the hair cells and/or an increase of the number of contributing hair cells The horizontal shift suggests that the sound transmission to the site within the organ of Corti where the nonlinearity of the intensity function of CM ensues is gradually improved

II Summating potentials (SP) and CM were recorded from scala media of the basal turn, usually in response to a standard type of tone burst with a frequency of 3 kc, an intensity of 125 db and a duration of 8 msec Under these conditions SP and CM were observed to appear at an age of 5 days The early type of SP consisted of a rapid negative potential shift followed by a slower increase of the negativity This negative SP was particularly prominent in animals 5 to 7 days old With further increase in age the negative SP decreased in magnitude and finally at an age of 15 days SP was found to be positive Also when recorded at the round window the polarity of SP was observed to change from negative to positive during development Under asphyxia the early type of negative SP gradually decreased whereas the positive SP recorded by the end of the development temporarily turned strongly negative

The findings are discussed in relation to previous observations on SP in adult guinea pigs In connection with reports from the literature that the development of the internal hair cells precedes that of the external ones, it is noted that the change of the polarity of SP during the development is consistent with previously reported observations on SP, which indicate that the internal hair cells contribute to the recorded SP with a negative component and the external hair cells with a positive component

III The endocochlear potential (EP) of the basal turn was found to appear at an age of 5 days During the following five days a rapid increase of EP was observed and subsequently at an age of about 15 days EP was found to have an adult value of about 80 mV In the middle and apical turns EP increased during this period in an approximately linear relation to EP in the basal turn In stria vascularis negative DC potentials were observed already before the appearance of EP and were subsequently found to increase in connection with the development of EP Under asphyxia EP was regularly found to turn negative

VI During the early phases of the functional development it was found difficult to record the neural component of the cochlear response with conventional methods From the age of 5 days a triphasic potential with an initial positive component could regularly be recorded at the trapezoid body with stereotactic technique, when stimulating with the standard 3 kc tone burst During development the latency of the positive peak decreased from 3-4 5 msec at 5 days and attained a final value of about 1 5 msec at 15 days The latency of the following negative peak decreased from 4 5 7 msec to 2 5 3 msec during the same period

Before the age of 8 days, animals which readily responded to the tone burst failed to respond when stimulated with an intense rarefaction click From the age of 10 days the amplitude of the potentials recorded in response to the two types of stimuli were of the same order of magnitude

The difference in stimulating efficiency between the two types of stimuli could be accounted for by their different durations In 5-day animals a 3 kc signal had to consist of at least 2 complete cycles in order to evoke a response On further increase of the duration of the signal up to 5 cycles the amplitude of the response increased At an age of 15 days the amplitude of the response was found to be unaffected by changes of the duration of the signal

It is concluded that the auditory nerve fibres at the time of appearance of the receptor potentials are ready to transmit nerve impulses During the subsequent development the efficiency of generation and transmission of impulses is gradually improved

V From the age of 5 days acoustically evoked contractions of the middle ear muscles were observed

VI The functional development is discussed in relation to the morphological differentiation of the inner ear as it appears from light microscopic studies performed in connection with the present study and from descriptions in the literature

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# BONE CONDUCTION AND NOISE MASKING

BY

PETER B. WESTON

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM O

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SUPPLEMENTUM 204

# BONE CONDUCTION AND NOISE MASKING

BY

PETER B. WESTON

UPPSALA 1965

# FOREWORD

The investigations reported in this monograph are based on a thesis entitled Bone conducted tones masked by air conducted noise which was presented in partial fulfillment of the requirements for the degree of Master of Arts Washington University St Louis Missouri August 1964

The author wishes to acknowledge the assistance and advice given during the course of these investigations by the staff of Central Institute for the Deaf In particular the author is indebted to Dr James D Miller who originally suggested the method described in this monograph for the determination of monaural masked thresholds for bone-conducted signals for his encouragement guidance and assistance throughout the main course of these investigations Drs Ira J Hirsh and Jerome R Cox Jr were responsible for the initiation and preliminary guidance of the project

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# INTRODUCTION

When the head of a normally hearing listener is vibrated by a bone-conduction transducer, both ears are stimulated to approximately the same extent because acoustic energy is transmitted through the whole skull (Barany, 1938, Bekesy, 1932) But bone-conduction audiometry is concerned primarily with the auditory sensitivity of one ear rather than both ears

In order to determine the auditory sensitivity of one ear to bone-conducted tones or signals, a common audiometric procedure is to mask the bone-conducted signal stimulating the other ear by means of air-conducted noise However, there are no quantitative data on exactly how air-conducted noise does, in fact, mask a signal that is introduced to a single ear by bone conduction

## THE METHOD

The primary purpose of this study was to develop and evaluate a method for obtaining monaural thresholds for bone-conducted signals masked by air conducted noises Such an experiment would be simple if persons with one "normal' ear, one "dead' ear, and a normal skull could be obtained However, such listeners are not readily available

Since the two ears of normal listeners are stimulated nearly identically by bone conduction, a method for determining thresholds for bone-conducted signals masked by air-conducted noise at one ear requires the functional elimination of the non-test ear In this study the elimination of the non test ear was accomplished by means of air-conducted noise In order to identify clearly the two air-conducted noises, the noise at the ear for which thresholds are to be determined is called masking noise, while the noise at the ear to be eliminated is called blocking noise

Two factors concerning the relations between the air-conducted noises and the bone-conducted signals must be considered One factor is the difference in level between the masking and blocking noises required to eliminate functionally or 'block the signal from the non-test ear The other is the possible effect of the blocking noise on the masked threshold for the bone-conducted signal at the test ear These factors were investigated for air-conducted signals by Weston and Miller (1965), and since their conditions were designed to be analogous to those for bone-conducted signals, their results are relevant here Their results strongly suggest the following: (1) one ear can be effectively eliminated from a masking experiment by

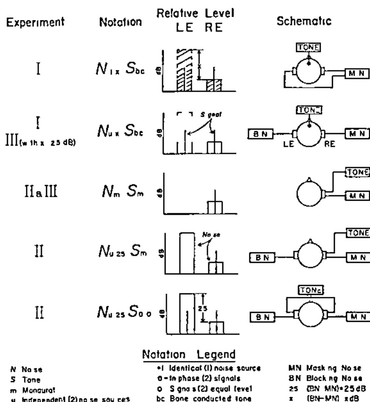



FIG 1 Notation for the experimental conditions The conditions used in each of the three experiments are shown

positive correlation of plus one or they were statistically independent (uncorrelated))

Experiment II was conducted to test the hypothesis that the masked thresholds for air conduction are identical for the following three conditions (1) the usual monaural masked threshold with both signal and noise delivered to the same ear, (2) same as (1) but an independent noise added to the opposite ear at a level 25 dB greater than the masking noise, and (3) same as (2) but with an additional signal, identical in all respects to the original one, introduced into the opposite ear Since the hypothesis was confirmed by the experiment, the air-conduction thresholds that were used for comparison with the bone-conduction thresholds in Experiment III were the usual monaural ones, i e condition (1)

Experiment III made extensive use of the method for measuring the masking of bone conducted signals by air-conducted noises in a single ear The results of Experiment III are described under the following six headings (1) The masking functions, (2) The occlusion effect, (3) Inconsistencies in threshold data (4) Physical measures of the threshold signals, (5) Possible applications of methods and results, and (6) Variability of thresholds

a blocking noise that is about 25 dB higher in level than the masking noise and (2) the masked threshold in the test ear is not influenced by the blocking noise if the blocking and masking noises are statistically independent

*Level differences between blocking and masking noises* The following argument supports the case that monaural masked thresholds can be determined for bone-conducted signals provided the blocking noise is at least 25 dB higher in level than the masking noise Signals introduced by bone conduction are about equal in level at the ears of normally hearing listeners When the listener adjusts the signal to threshold in the ear receiving the masking noise then the signal in the non test ear would be about 25 dB below its own monaural threshold This of course would be true since the blocking noise in the non test ear is 25 dB higher than the masking noise Since serveral lines of evidence (Weston and Miller, 1965 and Egan 1964) support the notion that a tone which is 25 dB below its monaural threshold has almost no effect on the auditory system it seems safe to assume that the effect of the signal in the non test ear has been removed from the experiment

*Correlation between blocking and masking noises* It can also be reasoned that the blocking and masking noises must be independent of each other that is derived from independent noise generators The reason for this requirement can be understood if one considers the case where the blocking and masking noises are from the same noise generator In this case the noises at the two ears would be identical in all respects but amplitude Now if the level of the blocking noise be 25 dB greater than that of the masking noise the signal would be effectively blocked from the non test ear but the noise would nonetheless by itself influence the masked threshold in the test ear Indeed the results of Weston and Miller (1965) and those of Egan (1964) show such effects even if the noise levels differ by as much as 40 dB If however the noise used as a blocking noise is statistically independent *of the masking noise little or no effect of the blocking* noise is observed on the masked threshold in the test ear

## The Experiments

The three experiments to be reported here investigate the adequacy and usefulness of a method for measuring the monaural masked threshold for bone-conducted signals

Experiment I shows that for bone conducted signals as was the case for air conduction (Weston and Miller 1965) the blocking noise must be both independent of and about 25 dB greater than the masking noise if the masked threshold is to be monaural Two major variables were investigated one the difference in level between the blocking and masking noises and the other the correlation between them (that is the noises had either a

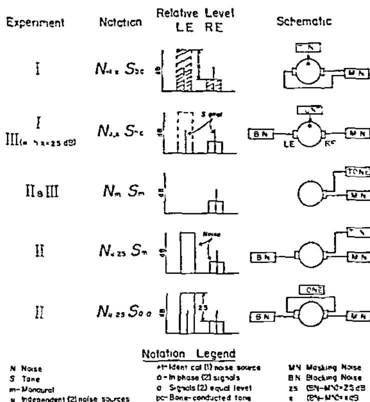



FIG 1 Notation for the experimental conditions The conditions used in each of the three experiments are shown

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Experiment II was conducted to test the hypothesis that the masked thresholds for air conduction are identical for the following three conditions (1) the usual monaural masked threshold with both signal and noise delivered to the same ear, (2) same as (1) but an independent noise added to the opposite ear at a level 25 dB greater than the masking noise, and (3) same as (2) but with an additional signal, identical in all respects to the original one, introduced into the opposite ear Since the hypothesis was confirmed by the experiment, the air-conduction thresholds that were used for comparison with the bone-conduction thresholds in Experiment III were the usual monaural ones, i e condition (1)

Experiment III made extensive use of the method for measuring the masking of bone-conducted signals by air conducted noises in a single ear The results of Experiment III are described under the following six headings (1) The masking functions, (2) The occlusion effect, (3) Inconsistencies in threshold data, (4) Physical measures of the threshold signals, (5) Possible applications of methods and results, and (6) Variability of thresholds

## Notation

In these experiments there are several complicated combinations of signals and noises at the two ears. A notation and corresponding descriptions of these conditions are given below and the same notation is illustrated by diagrams in Fig. 1.

The notation, although somewhat different from that used by others for the description of tonal signals and noises at the ears, allows for the expression of the difference in levels at the two ears. The first subscript indicates the correlation or phase between the noises or signals at the two ears if both ears are stimulated. A plus one stands for a positive and perfect correlation, while a *u* stands for uncorrelated or statistically independent noises. A zero indicates that the signals are in phase. If the first subscript is the letter *m*, the experiment was monaural. The letters *bc* indicate that the tonal signal is presented by bone conduction. An *x* for a second subscript means that the difference in levels between ears is a variable; if this second subscript is a number, the number is the level of the blocking noise minus the level of the masking noise in decibels. The illustrations show the blocking noise (BN) at the left ear and the masking noise (MN) at the right.

The conditions shown in Fig. 1 are from top to bottom as follows:

(1) $N_{1,x}\ S_{bc}$ — The noises are identical at the two ears in all respects except amplitude and $x$ is the level of the blocking noise (BN) minus the level of the masking noise (MN) in decibels, or $(BN - MN) = x$ dB. The signal is presented by bone conduction.

(2) $N_{u,x}\ S_{bc}$ — This condition is the same as (1) above but the noises at the two ears are independent.

(3) $N_m\ S_m$ — Signal and noise are presented to the right ear by air conduction.

(4) $N_{u,25}\ S_m$ — The noises are independent at the two ears and $(BN - MN) = +25$ dB. The signal is presented to the right ear by air conduction.

(5) $N_{u,25}\ S_{0,0}$ — This condition is the same as (4) above but the signals are in phase and of equal amplitude at the two ears.

# II. METHODS

## Apparatus

The essential features of the apparatus used in these experiments are briefly described in the text below and shown in Fig 7 of Appendix A A complete description is given Appendix A

*Block diagram* Basically, the apparatus can be thought of as having three channels one for the left earphone, one for the right earphone, and one for the bone-conduction transducer By appropriate combinations of switch positions, noise can be presented to either earphone from a single noise generator or from independent noise generators, and tone pulses can be routed to either earphone, both earphones, or the bone conduction transducer

*Bone-conduction transducer* A commercially available dynamic type bone-conduction transducer was modified and used in this study Two measurement systems were used for the calibration of the bone-conduction transducer One of the systems was also used to monitor the dynamic behavior of the transducer during the experiments Measurements of the response of the transducer were obtained in terms of rms velocity Thus physical measures could be calculated for bone conducted signals at threshold intensity Calibrations in terms of rms velocity were obtained with the transducer coupled to the frontal bone site of each subject

*Earphones* Two types of earphones or receivers were used in this study One type was a matched pair of dynamic earphones (Telephonics, Model TDH-39) mounted in small neoprene cushions (MX-41/AR) These receivers are commonly used in experimental and clinical studies The other type was a pair of receivers manufactured by M P Pedersen, Copenhagen, Denmark[1] Each of these receivers consists of a dynamic loudspeaker, seven inches in diameter, mounted in a metal sphere The loudspeaker is supported within the sphere by means of felt padding Soft rubber cushions are mounted on the spheres for circumaural contact with the head These receivers are mounted on an adjustable crossbar and tripod pedestal

In order to identify clearly the two types of receivers in the experiments to be reported, the dynamic earphones (Telephonics) will be called "Con-

[1] These receivers are on loan from Dr Barry S Elpern University of Chicago Clinics, Chicago, Illinois

## Notation

In these experiments there are several complicated combinations of signals and noises at the two ears. A notation and corresponding descriptions of these conditions are given below and the same notation is illustrated by diagrams in Fig. 1.

The notation, although somewhat different from that used by others for the description of tonal signals and noises at the ears, allows for the expression of the difference in levels at the two ears. The first subscript indicates the correlation or phase between the noises or signals at the two ears if both ears are stimulated. A plus one stands for a positive and perfect correlation, while a $u$ stands for uncorrelated or statistically independent noises. A zero indicates that the signals are in phase. If the first subscript is the letter $m$, the experiment was monaural. The letters $bc$ indicate that the tonal signal is presented by bone conduction. An $x$ for a second subscript means that the difference in levels between ears is a variable; if this second subscript is a number, the number is the level of the blocking noise minus the level of the masking noise in decibels. The illustrations show the blocking noise (BN) at the left ear and the masking noise (MN) at the right.

The conditions shown in Fig. 1 are from top to bottom as follows:

(1) $N_{+1,x}$ $S_{bc}$. The noises are identical at the two ears in all respects except amplitude and $x$ is the level of the blocking noise (BN) minus the level of the masking noise (MN) in decibels, or (BN − MN) = $x$ dB. The signal is presented by bone conduction.

(2) $N_{u\,x}$ $S_{bc}$. This condition is the same as (1) above but the noises at the two ears are independent.

(3) $N_m$ $S_m$. Signal and noise are presented to the right ear by air conduction.

(4) $N_{u,25}$ $S_m$. The noises are independent at the two ears and (BN − MN) = +25 dB. The signal is presented to the right ear by air conduction.

(5) $N_{u\,25}$ $S_{0\,0}$. This condition is the same as (4) above but the signals are in phase and of equal amplitude at the two ears.

# III. EXPERIMENT I

## *Masking of bone-conducted signals as related to level difference and the correlation between blocking and masking noises*

Experiment I was conducted to evaluate the method for obtaining monaural thresholds for bone-conducted signals masked by air-conducted noises The method, it will be recalled, required a masking noise at the ear at which thresholds for bone-conducted signals are to be determined and a blocking noise at the ear at which the effects of the bone-conducted signals are to be eliminated

As described in the Introduction, the results of the study by Weston and Miller (1965) for air-conducted 500-cps signals suggested that the blocking noise must be both independent of and about 25 dB higher than the masking noise in order to determine monaural masked thresholds at the ear receiving the masking noise Therefore, this experiment was designed to firmly establish the fact that the relations between the blocking and masking noises necessary for the determination of monaural thresholds for air-conducted 500-cps signals also hold for the case of bone conducted 500-cps signals as well as for bone conducted signals of other frequencies Thus, signals were delivered by bone conduction, while masking and blocking noises were delivered by air conduction

The design of the experiment included two variables (1) the difference in level between the blocking and masking noises and (2) the correlation between these noises, that is, the noises had either a positive correlation of plus one or they were statistically independent (uncorrelated) In the notation described in Chapter I and illustrated in Fig 1, these conditions were $N_{+1x}S_{bc}$ and $N_{ux}S_{bc}$

### *Specific Methods*

Although many of the procedures used in this experiment have been previously described (see Chapter I, General Procedure), certain aspects of the procedure were unique to this experiment

One block of the experimental design is shown in Table I A block consisted of four units, one for each frequency Two stimulus conditions, $N_{ux}S_{bc}$ and $N_{+1x}S_{bc}$, were used and they are given in column 3 of Table I Column 4 indicates the number of values of $x$ per condition, column 5 the

The difference in thresholds for these signals is nearly the same for all levels of the blocking noise The difference in thresholds for the 1000-cps signal is smaller and is a function of the level of the blocking noise, while for the 2000-cps signal the difference is almost nil

The curves shown in Fig 2 for the 500-, 1000-, and 2000 cps signals are based on "corrected" levels for the blocking noise The receiver that produced the blocking noise was found to have a hole in its diaphragm, and the data may be contaminated by distortion The curve for the 250-cps signal represents data obtained using a new set of matched receivers and these data are "clean"

## *Conclusions*

The curves shown in Fig 2, the results of Weston and Miller (1965), and the results of Experiment II to be reported in the next chapter, all support the notion that the determination of monaural masked thresholds for bone-conducted signals requires a contralateral blocking noise that is both independent of and about 25 dB higher in level than the masking noise

## A Further Comment

***Are both ears stimulated identically by the bone conduction transducer at the frontal bone site?***

A further comment concerns a general feature of the curves in Fig 2 Consider the curve for the correlated noises ($N_{+1x}$ $S_{bc}$), for the 500-cps signal As the blocking-noise level increases, the threshold gradually *rises* to a plateau Now Weston and Miller (1965) did a similar experiment, but their binaural signals were delivered by air conduction Those air conducted signals were known to be identical in all respects at the two ears In that case, the thresholds values gradually *decreased* to a plateau This difference in direction of the curve in Experiment I and in that of Weston and Miller (1965) casts doubt on the assumption that the effects of a bone conducted signal are identical at the two ears

It is not possible to determine from the present experiment whether the bone conducted signals at the two ears differ in amplitude, phase, or both, nonetheless, a phase difference between the ears of about 60° would account for these data (Durlach, 1963)

# IV EXPERIMENT II

## *Influence of signal and blocking noise in the non test ear on the masked threshold in the test ear*

Experiment II was conducted to ascertain which of three possible procedures for air conduction thresholds would be most appropriate for comparison with the monaural masked thresholds for bone-conducted signals the experiment shows the relations between the three air-conduction procedures

The conditions of the experiment were as follows (1) the conventional masked threshold obtained with both signal and noise delivered to the right ear ($N_m S_m$) (2) same as (1) but an independent noise added to the left ear at a level 25 dB higher than the original noise ($N_{m\,25} S_m$) and (3) same as (2) but an additional signal presented to the left ear and the new signal maintained identical in all respects to the signal in the right ear ($N_{m\,25} S_{0\,0}$) All signals were delivered by air conduction

### *Specific Methods*

Several of the procedures used in this experiment were described previously (see Chapter II General Procedure) but certain aspects of the procedure were unique to this experiment Four subjects were used A single block of the experiment consisted of four units one for each frequency Within each unit two threshold determinations were obtained for each of the three conditions Each subject completed one experimental block during a sitting on five occasions Thus there were five replications for each experimental block and therefore 40 threshold determinations were obtained for each combination of condition and frequency The order of the units within a block and the order of the conditions within a unit were separately randomized Each unit began with a determination of the quiet threshold for each ear then the three noise conditions were run and finally the determinations of the quiet thresholds were repeated

The Conventional Receivers (TDH 39 dynamic earphones mounted in MX 41 AR cushions) were used in this experiment The spectrum levels (re 0 0002 μbar for the masking noises were +29 7 +19 7 +13 9 and +11 8 dB for the signal frequencies of 250 500 1000 and 2000 cps respectively The spectrum levels of the blocking noises for the binaural noise conditions were 25 dB higher in level than those of the masking noises

TABLE II *Results of Experiment II for the three conditions in terms of sound pressure level (SPL) according to frequency (cps)*

The differences in decibels between the monaural condition ($N_m S_m$) and the two binaural conditions are also shown

| | 250 | | 500 | | 1000 | | 2000 | |
|---|---|---|---|---|---|---|---|---|
| Conditions | SPL | Diff | SPL | Diff | SPL | Diff | SPL | Diff |
| $N_m S_m$ | 48 0 | | 37 5 | | 32 2 | | 30 8 | |
| $N_{u,25} S_m$ | 47 8 | −0 2 | 36 6 | −0 9 | 31 5 | −0 7 | 30 7 | −0 1 |
| $N_{u,25} S_{00}$ | 47 2 | −0 8 | 37 0 | −0 5 | 31 7 | −0 5 | 30 9 | +0 1 |

## *Results*

Table II shows the results for each of the three conditions The results are each the mean of 40 threshold determinations and they are expressed in sound pressure levels (SPL's) The differences between the thresholds obtained for the monaural noise condition, $N_m S_m$, and those obtained for each of the two binaural noise conditions, $N_{u\,25} S_m$ and $N_{u\,25} S_{0\,0}$, are also shown The mean difference between the thresholds for the condition $N_m S_m$ and those for the condition $N_{u\,25} S_m$ is −0 475 dB, while the mean difference between the thresholds for the condition $N_m S_m$ and those for the condition $N_{u\,25} S_{0\,0}$ is −0 425 dB All of the differences (except for one condition at 2000 cps) fall in the same direction This indicates that the two binaural conditions produce a very small (about 0 5 dB) change in the masked threshold It should be noted that in the case of the condition $N_{u\,25} S_{0\,0}$ the signal at the left ear has no effect on the threshold Thus, one can infer that the threshold was obtained for the signal at the right ear

## *Discussion and Conclusions*

These results strongly support those of Weston and Miller (1965), which were obtained for a 500-cps signal, and they provide a firm basis for extending their conclusions to other frequencies Thus, it appears that a noise in one ear has little or no effect on the masked threshold for a signal in the other ear if these noises are independent, that is, from separate noise generators This is true even if the noise in the ear which does not receive the signal, that is the blocking noise, is 25 dB higher in level than the masking noise in the ear which does receive the signal Further, if signals identical in all respects are delivered to the two ears, the one presented to the ear receiving the blocking noise has no effect on the threshold for the signal at the other ear

These facts provide additional support for the rationale behind the method to be used in Experiment III to obtain monaural thresholds for bone conducted signals masked by air conducted noise. Moreover, the results of the present experiment show that the signal thresholds for the three conditions which were tested are nearly the same and therefore one may measure such thresholds using the most convenient or simple procedure.

## A Further Comment

### *Right versus left ear*

Possibly the conclusions given in the previous section are correct only if the blocking noise is presented to the left ear while the masking noise is presented to the right ear. However, it seems unlikely that the results would differ if the blocking noise were presented to the right ear and the masking noise were presented to the left ear, but the possibility must be noted for the unwary.

# V. EXPERIMENT III

## *Monaural thresholds for bone-conducted and air-conducted signals masked by air-conducted noises*

### *Introduction*

This experiment made extensive use of the method for measuring monaural thresholds for bone-conducted and air conducted signals masked by air-conducted noises Masked thresholds for the air-conducted signals were obtained by the usual monaural procedure ($N_m$ $S_m$); while the masked thresholds for the bone-conducted signals were obtained using independent blocking and masking noises with the blocking noise 25 dB higher in level than the masking noise ($N_{B\,25}$ $S_{bc}$) The experiment was designed to provide the kinds of information classified and described below

(1) *The masking functions* Curves relating the monaural masked threshold to noise level are presented for both bone- and air-conducted signals, these functions are determined at 250, 500, 1000, and 2000 cps

(2) *The occlusion effect* This effect is assessed at both masked and quiet threshold

(3) *Physical measures of threshold signals* Since the air- and bone-conduction transducers were calibrated in physical units, physical measures could be calculated for both types of signals at threshold intensity in either noise or quiet

(4) *Variability of thresholds* Measures of the variability of the thresholds are presented for bone- and air-conducted signals in quiet and in noise

Possible applications of the methods and results to two practical problems are discussed One problem is that of estimating the amount of air-conducted noise required to mask a bone conducted tonal signal, the other is that of the "real-head" calibration of bone-conduction transducers at high as well as low output levels

### *Specific Methods*

In this experiment the electronic apparatus shown in Fig 7 in Appendix A was used in conjunction with the Conventional and Pedersen Receivers

The bone-conduction thresholds were measured in decibels of attenuation (with 0 dB = 3 16 volts across the bone-conduction transducer's terminals) f l b i conducted signal and were later transformed to rms velocities

**Table III** *One block of the experimental design for the Conventional Receivers for Experiment III*

See text for a full description

| Units | Frequency (cps) | Signal | Condition | Thresholds per condition | Thresholds per unit | Masking noise spectrum level |
|---|---|---|---|---|---|---|
| 1 | 250 | Bone | QAT[a] | 2 | 10 | OFF (BN = +29)[b] |
| | | | $N_{u,25}\ S_{bc}$ | 2 | | +20, −30, −40, −50 |
| | | Air | QAT | 2 | 6 | OFF |
| | | | $N_m\ S_m$ | 2 | | −30, +50 |
| 2 | 500 | Bone | QAT | 2 | 10 | OFF (BN = +19) |
| | | | $N_{u,25}\ S_{bc}$ | 2 | | +10, +20, +30, +40 |
| | | Air | QAT | 2 | 6 | OFF |
| | | | $N_m\ S_m$ | 2 | | +20, +40 |
| 3 | 1000 | Bone | QAT | 2 | 10 | OFF (BN = +8) |
| | | | $N_{u,25}\ S_{bc}$ | 2 | | +4 +14, 24 +34 |
| | | Air | QAT | 2 | 6 | OFF |
| | | | $N_m\ S_m$ | 2 | | +14, +34 |
| 4 | 2000 | Bone | QAT | 2 | 10 | OFF (BN = +7) |
| | | | $N_{u,25}\ S_{bc}$ | 2 | | +2, +12, +22, +32 |
| | | Air | QAT | 2 | 6 | OFF |
| | | | $N_m\ S_m$ | 2 | | +12 +32 |

[a] QAT stands for quiet absolute threshold which was determined for each ear

[b] Indicates the blocking noise (BN) level used for QAT determinations at the other ear

Velocity calibrations were carried out with the bone-conduction transducer coupled to the subject's frontal-bone site with 750-grams force A continuous signal was fed to the transducer at 3 16 volts (with the subject's and experimenter's attenuators both set for zero attenuation) and the output of the pickup system (as described in Appendix A) was measured in decibels (re 1 0 volt) The pickup system was adjusted so that an output of 1 0 volt was obtained for an input of 1 0 cm/sec rms Thus, velocity measurements in dB[1] were obtained from the pickup system for a constant voltage at the input of the transducer The actual velocity in decibels of the signal present at threshold was simply the calibration measurement minus the total attenuation introduced by the subject's and experimenter's attenuators

The air-conduction thresholds were measured in decibels of attenuation (with 0 dB = 0 1 volt across the receiver's terminals) and were later transformed to sound pressure levels (SPL's) by means of the calibrations for the two sets of receivers as shown in Table IX in Appendix A

[1] Velocity in dB is defined as dB = 20 log [(cm/sec)/(ref )], where the reference is 1 0 cm sec (rms)

TABLE IV *One block of the experimental design for the Pedersen Receivers for Experiment III*

See text for a full description

| Units | Frequency (cps) | Signal | Condition | Thresholds per condition | Thresholds per unit | Masking noise spectrum level |
|---|---|---|---|---|---|---|
| 1 | 250 | Bone | QAT[a] | 2 | 10 | OFF (BN = +35)[b] |
| | | | $N_{u,25} S_{bc}$ | 2 | | +21, +26, +31, +36 |
| | | Air | QAT | 2 | 6 | OFF |
| | | | $N_m S_m$ | 2 | | +26, +36 |
| 2 | 500 | Bone | QAT | 2 | 10 | OFF (BN = +24) |
| | | | $N_{u\,25} S_{bc}$ | 2 | | +13, +18, +23, +28 |
| | | Air | QAT | 2 | 6 | OFF |
| | | | $N_m S_m$ | 2 | | +18, +28 |
| 3 | 1000 | Bone | QAT | 2 | 10 | OFF (BN = +16) |
| | | | $N_{u\,25} S_{bc}$ | 2 | | +1, +11, +21, +31 |
| | | Air | QAT | 2 | 6 | OFF |
| | | | $N_m S_m$ | 2 | | +11, +31 |
| 4 | 2000 | Bone | QAT | 2 | 10 | OFF (BN = +29) |
| | | | $N_{u\,25} S_{bc}$ | 2 | | +3, +13 +23 |
| | | Air | QAT | 2 | 6 | OFF |
| | | | $N_m S_m$ | 2 | | +13, +23 |

[a] QAT stands for quiet absolute threshold which was determined for each ear

[b] Indicates the blocking noise (BN) level used for QAT determinations at the other ear

The general procedures used in this experiment have been described previously (see Chapter II, General Procedure), however, certain aspects of the procedure were peculiar to this experiment Each subject completed ten blocks, five for the Conventional Receivers and five for the Pedersen Receivers One block is shown in Table III for the Conventional Receivers and in Table IV for the Pedersen Receivers and the sequence in which the blocks were presented to each subject is given in Table V Each block was further subdivided into four units, one for each frequency During a single

TABLE V *Table of receiver sequence by replication for each subject in Experiment III*

| Replications | 1 | | 2 | | 3 | | 4 | | 5 | |
|---|---|---|---|---|---|---|---|---|---|---|
| Subjects 1 & 2 | CR[a] | PR[b] | PR | CR | CR | PR | PR | CR | CR | PR |
| Subjects 3 & 4 | PR | CR | CR | PR | PR | CR | CR | PR | PR | CR |

[a] CR stand [illegible] onventional Receiver

[b] PR stands Pedersen Receiver

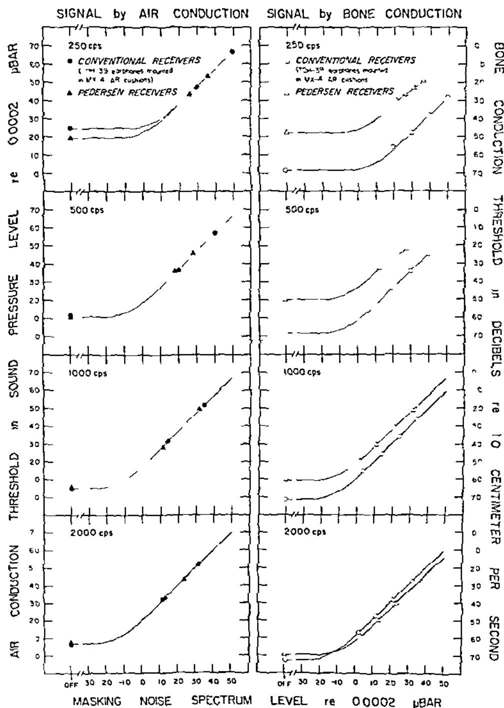



Fig. 3. The results for Experiment III. The masking functions are shown for both air conduction ($N_m S_m$) and bone conduction ($N_m S_b$) as obtained for the Conventional and Pedersen Receivers. See text for a complete description.

TABLE VI *Mean data of Experiment III*

Quiet (noise OFF) and masked thresholds are shown as a function of signal frequency for the Conventional and Pedersen Receivers Air-conduction thresholds are in sound pressure level (SPL) re 0.0002 $\mu$bar, while bone-conduction thresholds are in velocity expressed in decibels (dB) re 1.0 cm/sec (rms)

| | Conventional Receivers | | | Pedersen Receivers | | |
|---|---|---|---|---|---|---|
| Frequency (cps) | Masking noise spectrum level | Bone conduction dB re 1 cm/sec | Air conduction SPL | Masking noise spectrum level | Bone conduction dB re 1 cm/sec | Air conduction SPL |
| 250 | OFF | −68.4 | +24.6 | OFF | −47.6 | +19.0 |
| | +19.7 | −55.8 | — | +21.1 | −30.4 | — |
| | +29.7 | −47.8 | +46.8 | +26.1 | −26.9 | +43.1 |
| | +39.7 | −38.1 | — | +31.1 | −23.1 | — |
| | +49.7 | −28.2 | +66.6 | +36.1 | −19.4 | +53.3 |
| 500 | OFF | −68.4 | +12.0 | OFF | −50.0 | +10.6 |
| | +9.7 | −53.0 | — | +12.9 | −32.2 | — |
| | +19.7 | −44.1 | +36.9 | +17.9 | −27.8 | +35.7 |
| | +29.7 | −34.1 | — | +22.9 | −25.2 | — |
| | +39.7 | −24.3 | +57.2 | +27.9 | −21.9 | +45.3 |
| 1000 | OFF | −71.5 | +4.9 | OFF | −60.7 | +5.8 |
| | +3.9 | −55.4 | — | +1.2 | −49.8 | — |
| | +13.9 | −45.6 | +31.7 | +11.2 | −40.8 | +27.9 |
| | +23.9 | −36.0 | — | +21.2 | −31.0 | — |
| | +33.9 | −26.2 | +51.7 | +31.2 | −21.8 | +49.1 |
| 2000 | OFF | −72.7 | +6.0 | OFF | −69.8 | +7.9 |
| | +1.8 | −56.9 | — | +3.3 | −59.3 | — |
| | +11.8 | −47.1 | +32.0 | +13.3 | −49.9 | +33.1 |
| | +21.8 | −38.0 | — | +23.3 | −40.1 | +43.4 |
| | +31.8 | −27.5 | +52.0 | — | — | — |

block, thresholds were determined for each unit in the quiet and in noise with the signal delivered by bone and air conduction For each block the order of units and signal pathways (bone or air conduction) were separately randomized Further, for each condition within a unit the order in which the levels of the masking noise were presented was randomized

## *Results and Discussion*

### THE MASKING FUNCTIONS

*Mean results*

*The results of this experiment are shown in the eight panels of Fig 3* The abscissae for each of the eight panels are the spectrum levels (re

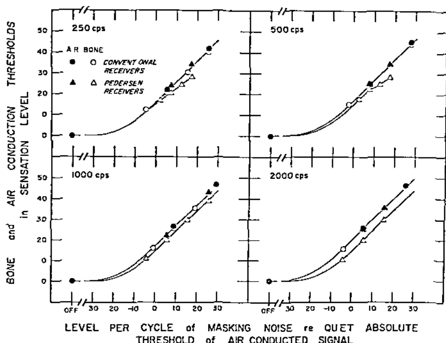



Fig 4 The data of Fig 3 replotted in sensation levels The levels per cycle of the masking noise are expressed relative to the quiet absolute thresholds of the test signals indicated in each panel

0 0002 $\mu$bar) of the masking noise The ordinates on the left of each panel are sound pressure levels (SPL s) of the air-conducted signals, while those on the right are rms velocities expressed in decibels re 1 0 cm/sec of the bone conducted signals The test signals are ordered in frequency (250, 500, 1000, and 2000 cps) from the top to the bottom panels The panels on the left show the data obtained for air conduction, while the panels on the right show the data obtained for bone conduction

The bone conduction thresholds, represented by the open circles for the Conventional Receivers (TDH-39 earphones mounted in MX-41/AR cushions) and by the open triangels for the Pedersen Receivers, are to be read from the vertical axis on the right of each panel The air-conduction thresholds, represented by the filled circles for the Conventional Receivers and by the filled triangles for the Pedersen Receivers, are to be read from the vertical axis on the left of each panel On each panel the point above the abscissa marked 'OFF' is a quiet absolute threshold

The results shown in Fig 3 are the basic data of the experiment and they are given in numerical form in Table VI Each masked threshold represents the mean of 40 determinations, while each quiet threshold represents the mean of 20 determinations

The data of Fig 3 are replotted in Fig 4 For Fig 4 the abscissae for the four panels are the levels per cycle of the masking noises expressed

relative to the quiet absolute threshold for each of the signal frequencies The ordinates for the panels are sensation levels (SL's). The key which matches the data points to the experimental conditions is the same as in Fig 3

*Shapes of the masking functions*

The monaural masked threshold is related to the noise level by the data given in Table VI and shown in Fig 3 The curves shown in Figs 3 and 4 are called "masking functions" The data for the thresholds for the air-conducted signals were obtained in the quiet and at two levels of the masking noise Only these few points were needed because the masking functions for air conduction are well established in the literature (Hawkins and Stevens, 1950) On the other hand, four masked and a quiet threshold were obtained for each of the bone-conducted signals since these were essentially "new" experiments

*Linear cases* The shapes of fourteen of the sixteen curves in Fig 3 are highly similar and show the expected relation between noise level and the threshold for the tonal signal After an initial curvilinear section, they are linear with a slope of about one Indeed, fourteen of the sixteen functions shown in Fig 3 were fitted to the data by means of a "template curve" derived from the data of Hawkins and Stevens (1950) These results, with the exceptions to be discussed below, show that the functions relating the masked threshold to the level of air conducted noise, have similar shapes whether the tonal signal is delivered by bone or by air

*Non-linear cases* For more than about 10 dB of masking, all of the masking functions were linear with slope one, except those for bone-conducted signals of 250 and 500 cps obtained when the receivers with large volume (that is, the Pedersen Receivers) were over the ears These non-linear functions can be observed in the various plots of the data shown in Figs 3 and 4

It may be that the non-linear masking functions are related to activation of the intra-aural muscles by the blocking noise Since this hypothesis requires that the intra-aural muscles be activated by the blocking noise, this possibility was tested by measuring the change in the magnitude of the acoustic impedance at the eardrum when various noise levels were presented to the contralateral ear [1] It was found that the two highest levels of blocking noise used for the 250- and 500-cps tones (overall levels of 103 5- and 93 5-dB SPL for the Conventional Receivers and of 90 5- and 81 5 dB SPL for the Pedersen Receivers) definitely resulted in changes in the magnitude of the acoustic impedance at the test ear At lower levels of the blocking noise, the presence of such changes could not be detected with certainty These observations, plus consideration of the literature (in particular, Lilly,

[1] These measurements were performed by Dr David J Lilly

1964), support the notion that the blocking noise did, in fact, activate the intra-aural muscles

When the Pedersen Receivers are over the ears, there appears to be no occlusion effect (see later section) Thus, it is believed that the vibratory energy from the bone-conduction transducer is transmitted directly through the skin and skull to the labyrinths It is further hypothesized that contraction of the intra-aural muscles would attenuate the transmission of the air-conducted masking noise through the middle ear and, thus, lower the effective noise level at the basilar membrane The effect of the contraction of the intra aural muscles on the bone-conducted signal is not clear When the stapes is fixed, the bone-conducted energy delivered to the basilar membrane might be increased or decreased depending on the particular bone-conduction mechanism that predominates Non-linear masking functions of the type observed would occur if the activation of the intra aural muscles attenuated the air-conducted noise more than it attenuated the bone-conducted signal or if it attenuated the air-conducted noise and enhanced the bone-conducted signal

When the Conventional Receivers were used, the masking functions were linear and, yet, the intra aural muscles were probably activated This apparent conflict can be explained in the following way When the Conventional Receivers were over the ears, the well-known occlusion effect is observed (see later section) Although several explanations of the occlusion effect have been offered, Békésy (1932), Barany (1938), Zwislocki (1953), Allen and Fernandez (1960), and Tonndorf (1964), the crucial hypothesis for the present argument is that the vibratory energy from the bone-conduction transducer produces an acoustic signal in the external auditory meatus, and that this acoustic signal in the external auditory meatus is so large that it controls the threshold If these assumptions be true, then attenuation of transmission through the middle ear by activation of the intra aural muscles would result in equal attenuation to both the air-conducted noise and the signal resulting from the bone-conduction transducer If both signal and noise are reduced equally, the masking functions would be linear as was found here

In addition to the theory presented above, certain observations made by the subjects may be of significance They all reported difficulty in determining the masked thresholds for bone conduction when the signal was 250 or 500 cps, but only when the Pedersen Receivers and not when the Conventional Receivers were used The comments were that it was difficult to differentiate between "feeling" and "hearing" the signal when it was set to threshold intensity It is not known how to interpret these comments

## The Occlusion Effect

The experiment was designed to assess the "occlusion effect" on the masked as well as the quiet threshold of bone-conducted signals The oc-

clusion effect is the lowering of bone-conduction thresholds at low frequencies when the external ears are occluded. Different degrees of occlusion were provided, in this experiment, by two kinds of earphones. The Conventional Receivers (TDH-39 dynamic earphones mounted in MX-41/AR cushions) occlude with only a small volume and produce a sizable occlusion effect, while the Pedersen Receivers, with a large volume, produce little or no occlusion effect (Elpern and Naunton, 1963).

The magnitude of the occlusion effect for the Conventional Receivers can be determined from the data in Table VI and from Fig. 3. The difference in thresholds between the two types of receivers is nearly the same at masked as it is at quiet threshold. The mean difference in thresholds for a 250 cps signal is about 22 dB, while for a 500 cps signal it is 18 dB. The occlusion effect for a 1000 cps signal is about 10 dB, while for a 2000 cps signal it is very small, that is, within ±3 dB.

## Inconsistencies in Threshold Data

If the bone-conducted signals delivered to the cochlea with the Conventional Receiver over the ears differed from those with the Pedersen Receivers over the ears by a constant amount, and if the thresholds for these two conditions differed for no other reason, it should be possible to "eliminate" the occlusion effect from the data by expressing the results in terms of masking, that is, masked threshold in dB minus quiet threshold in dB.

For tonal signals or noises delivered by air conduction, the two kinds of receivers might be equated in terms of their calibrations on couplers or in terms of the electrical power required at the quiet threshold for the air-conducted signals.

It was found that the two methods for equating the acoustic signals *generated by the two types of receivers did not agree perfectly, see Fig. 6* for more details. Therefore, it seems desirable to equate the noises produced by the two kinds of receivers in terms of the quiet thresholds for the relevant tones.

Figure 4 provides a test of the assumptions listed above excluding those concerning coupler calibrations. It can be seen that these assumptions are supported by the data except for the non linearities, previously discussed, and for the fact that the functions for bone-conducted signals with the Pedersen Receivers over the ears fall slightly below the others at 250, 500, and 1000 cps and about 5.5 dB below the others at 2000 cps. For reasons too uncertain and complicated to discuss here, it is believed that the 5.5 dB discrepancy at 2000 cps is due to a combination of errors in the measurement of the quiet thresholds at 2000 cps for both air-conducted and bone-conducted signals when the Pedersen Receivers were over the ears.

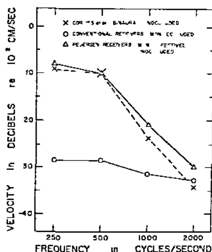



FIG 5 Quiet absolute thresholds for bone conduction obtained for the Conventional and Pedersen Receivers Data reported by Corliss *et al* (1959) are also shown

## PHYSICAL MEASURES OF THE THRESHOLD SIGNALS

### *Bone conduction*

The data for quiet absolute thresholds for bone-conducted signals with the Pedersen or Conventional Receivers over the ears are shown in Fig 5 by the triangles and circles, respectively Also shown are binaural thresholds obtained with the ears unoccluded as reported by Corliss, Smith, and Magruder (1959), although not shown the data of Bekesy (1932) and Watson (1938) are in good agreement with those of Corliss *et al* (1959)

The agreement between the present data with the Pedersen Receivers over the ears and those of Corliss *et al* (1959), Bekesy (1932), and Watson (1938), suggests that it is possible to specify the normal threshold for bone-conducted signals in terms of a physical measure of the output of the bone-conduction transducer Incidentally, the agreement adds further support to the conclusion of Elpern and Naunton (1963) that the Pedersen Receivers produce no "occlusion effect" The differences between the $x$'s and the triangles in Fig 5 of about 3 dB may be due to the differences between subjects, or differences between monaural and binaural thresholds, or they may result from the use of different measurement systems and techniques The difference between these data at 2000 cps may be due to the inadvertent use of an excessively high spectrum level of the blocking noise during threshold determinations [1]

[1] This situation arose because the Pedersen Receivers fall off sharply above 500 cps (see Appendix A) For this and several other reasons it is suggested that narrow band noise be used in future applications of this method

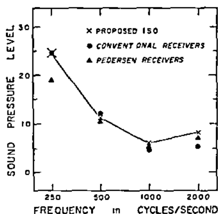



FIG 6 Quiet absolute thresholds for air conduction for the Conventional and Pedersen Receivers The proposed ISO thresholds are also shown

The data shown in Fig 5 for the Conventional Receivers show the lowering of threshold due to the occlusion effect, moreover, these thresholds are nearly independent of frequency and have a value of about −70 dB re 1 0 cm/sec (rms)

*Air conduction*

The data for quiet absolute thresholds for air-conducted signals obtained with the Conventional and the Pedersen Receivers are shown in Fig 6, along with data for the proposed ISO[1] standard for quiet absolute threshold (Davis and Kranz, 1964) For Fig 6 the abscissae are the signal frequencies, while the ordinates are sound pressure levels calculated from the coupler calibrations described in Appendix A The quiet thresholds are represented by the filled circles for the Conventional Receivers and the filled triangles for the Pedersen Receivers, while the *x*'s represent the thresholds for the proposed ISO standard

The agreement between threshold measurements for this experiment and the proposed ISO standard is good with the exception of the Pedersen Receivers at 250 cps The lower threshold obtained with the Pedersen Receivers at 250 cps cannot be accounted for in terms of the "missing 6 dB" as one might think This is so because the mean threshold for the Pedersen Receivers at 250 cps obtained from the left ears (not shown in Fig 6) is nearly a dB higher than the ISO threshold, while for the Conventional Receivers the mean thresholds are the same for the right and the left ears It seems likely that the difference in thresholds between the ears obtained with the Pedersen Receivers is due to unknown errors resulting from the coupler calibrations of these receivers

[1] ISO stands for the International Organization for Standardization

**Table VII** *Sound pressure levels in decibels (re 0.0002 µbar) for air conducted signals that are equivalent to bone-conducted signals generated when the velocity at the driver tip is 1.0 cm/sec (rms)*

The equivalence was determined at either masked or quiet threshold and extrapolated decibel for decibel, to the reference velocity of 1.0 cm/sec (rms)

| | Conventional Receivers | | Pedersen Receivers | |
|---|---|---|---|---|
| Frequency (cps) | Quiet SPL | Masked SPL | Quiet SPL | Masked SPL |
| 250 | 93.0 | 94.7 | 66.6 | 68.5(?)[a] |
| 500 | 80.4 | 81.3 | 60.6 | 63.0(?)[a] |
| 1000 | 76.4 | 77.6 | 66.5 | 69.4 |
| 2000 | 78.7 | 79.3 | 77.7 | 83.3[b] |

[a] The values for 250 and 500 cps were calculated from points believed to be on the linear portion of the masking function curves shown in Fig 3

[b] See the section on "Inconsistencies in Threshold Data" for a discussion of this deviant effect at 2000 cps for the Pedersen Receivers

### *Equivalence of physical measures*

Since stimulation of the inner ear is identical for both air- and bone-conduction pathways, for each experimental condition in this experiment (frequency, receiver, and kind of threshold) the physical measure for the bone conducted signal has an equivalent physical measure for the air-conducted signal when each signal is set to its respective threshold Table VII shows the equivalence between the physical measures for air and bone-conducted signals for the several conditions of this experiment The values given in Table VII were computed from the data given in Table VI according to the formula $x$-dB SPL $\equiv y$-dB velocity, where $x$ is the sound-pressure level (SPL) in decibels (re 0.0002 µbar) and $y$ is the velocity in decibels (re 1.0 cm/sec (rms)) of the driver tip of the bone-conduction transducer For this table $y$ is equal to 1.0 cm/sec (rms) and the values for $x$ are given for the several experimental conditions

## Possible Applications of Methods and Results

### *The determination of masking*

The masking functions for bone conduction shown in Fig 3 can be used to determine the amount of noise required to produce a specific amount of masking of a bone-conducted signal at one ear When the data in Fig 3 are used for these determinations the results are only correct for the air- and bone conduction transducer configurations used in this experiment.

However it is almost certain that similar data and masking functions could be obtained for other transducer configurations Similar functions could be interpolated for receivers that produce intermediate amounts of the occlusion effect

It should be emphasized that implicit in the determination of masking from the masking functions shown in Fig 3 is the fact that the blocking noise is always both independent of and 25 dB higher in level than the masking noise

Although the masking functions shown in Fig 3 are predictable from those for air-conducted signals the literature provided no direct information on this point The present experiment measures directly the amount of masking of a bone-conducted signal produced by an air conducted noise when both signal and noise are at the same ear Previous experiments have been designed to show whether too little or too much noise was applied to the non test ear (for example see Studebaker 1962 *a* or Konig 1963) and these experiments provided only indirect evidence about the masking of the bone-conducted signal at the non test ear

*Real head calibration of bone-conduction transducers*

The fact that most of the functions in Fig 3 are linear suggests that the bone-conduction transducer is operating within its linear range Non linear curves for 250 and 500 cps with the Pedersen Receivers over the ears are not traceable to the transducer and these non linear curves were discussed in a previous section

Since most of the masking functions for bone conduction are linear with slope one, that is a 10-dB increase in the level of the noise requires a 10-dB increase in the level of the signal to maintain threshold the method provides a means for the calibration of bone-conduction transducers at input levels greater than those required for the quiet threshold Another advantage of this technique is that the thresholds are under control of the experimentally introduced noise and thus the effects of ambient noises can be eliminated from the calibration

## Variability of Thresholds

*Introduction* The design of the experiment allowed the calculation of the variances and standard deviations of distributions of signal levels required at threshold These measures of threshold variability are described and discussed below

*Within listeners conditions and replications* ($\sigma_{\bar{w}_{LCR}}$) Since each listener adjusted the signal to masked threshold twice for each masked condition within a replication it was possible to calculate the standard deviations of the measures so obtained Variance estimates were averaged over test frequencies listeners and replications The square roots of these mean

TABLE VIII *Summary table of standard deviations of threshold signal in decibels*

| | Bone conduction | | | | Air conduction | | | |
|---|---|---|---|---|---|---|---|---|
| | Conventional | | Pedersen | | Conventional | | Pedersen | |
| | Quiet | Masked | Quiet | Masked | Quiet | Masked | Quiet | Masked |
| $\sigma_{W_{LCR}}$ | — | 1 220 | — | 1 313 | — | 1 195 | — | 1 242 |
| $\sigma_{W_{\overline{LCR}}}$ | — | 0 863 | — | 0 978 | — | 0 845 | — | 0 881 |
| $\sigma_{W_{LC}B_R}$ | 2 675 | 2 532 | 2 708 | 2 276 | 1 922 | 1 323 | 1 587 | 1 216 |
| $\sigma_{W_{CR}B_L}$ | 4 860 | 6 181 | 5 709 | 5 459 | 3 234 | 1 608 | 3 286 | 1 702 |

variances, that is the standard deviations are shown in the first row of Table VIII These standard deviations have a value of about 1 2 dB and show that the subject adjusts the signal to 'threshold' quite reliably within an experimental session Furthermore, there appears to be no difference between experimental conditions with respect to this kind of reliability

The standard deviation of 1 2 dB given above represents the variability of single measurements of the thresholds that is the irreducible error in a single, 20 sec determination of a masked threshold However, in the re mainder of the computations the basic datum used for the masked thresh old for each combination of listener and condition was the mean of the two measures of the threshold the standard deviations for these means were computed by the formula

$$\sigma_{W_{\overline{LCR}}} \quad \sigma^2_{W_{LCR}}/2]^{\frac{1}{2}}$$

and they are given in the second row of Table VIII Since the means of the two measures have standard deviations of less than 1 dB it is clear that the masked thresholds for either bone-conducted or air conducted signals were determined with a high level of reliability for each listener under each condition within each replication These standard deviations represent the variability in threshold measures that obtain when the subject is placed in the apparatus and neither the bone conduction transducer or the receivers are disturbed during the course of the session

*Within listeners and conditions between replications* ($\sigma_{W_{LC}B_R}$) The third line of Table VIII shows the standard deviations obtained when listeners and conditions are held constant and the data are analyzed for variation over replications A basic datum for a quiet threshold is one measure per replication while a basic datum for a masked threshold is the mean of two measures per replication It can be seen that the standard deviations of masked thresholds across replications are increased over those within replications by a factor of about 2 7 for bone-conducted signals and of about 1 4 for air conducted signals

It is possible to compare the variability of the threshold measures in the quiet with those in noise if allowance is made for the fact that the quiet thresholds are based on one measure, while the masked thresholds are means of two measures It appears that quiet and masked thresholds are about equally reliable

*Within conditions and replications, between listeners* ($\sigma_{W_{CR}B_L}$) The between listener standard deviations are given in the last row of Table VIII Individual differences in threshold can be ranked from largest to smallest as follows (1) bone-conducted signal in the air-conducted noise, (2) bone conducted signal in the quiet, (3) air-conducted signal in the quiet, and (4) air-conducted signal in air-conducted noise

*Variances for other categorizations of the data* Detailed results of the computations of variances and standard deviations of the threshold measures are given in Appendix B One additional fact seems to be that individual differences in the thresholds for bone-conducted signals are considerably larger at 1000 and 2000 cps than they are at 250 and 500 cps

*Summary and discussion of variability of thresholds* It appears that the coupling between the bone-conduction transducer and the subject's head cannot be reproduced from session to session with the same degree of accuracy as the placement of earphones over the ears

Indeed for a given subject, the variance in thresholds over experimental sittings is about four times greater for bone-conducted than for air-conducted signals while within a sitting no difference in reliability can be observed

When both signal and noise are delivered by air-conduction, the variance of thresholds both within and between subjects is small This is true since both the signal and the relevant band of noise are transmitted over almost identical channels right up to the mechanism in the brain that makes the decision, 'this is threshold '

For air-conducted signals the within listener–between replication variance is probably controlled by nearly the same variables in the quiet as it is in noise, although the variations in the coupling of the receiver to the head should be more important in the quiet than it is in the noise Differences among individuals should be greater in the quiet than they are in noise since all relevant properties of the auditory system can contribute to the between listener variance in the quiet, but the signal to noise ratio is the principal variable in the noise

For bone-conducted signals, the within listener–between replication variance is controlled primarily in both noise and quiet by the variations from sitting to sitting in the coupling of the bone-conduction transducer to the head In the noise however, variations from sitting to sitting of the air-conducted noise may contribute slightly to the variability of the thresholds

Individual differences in bone conducted thresholds are larger than individual differences in air conducted thresholds In the quiet this fact probably reflects uncontrolled differences between listeners with regard to both the fleshy and bony structure of their heads For bone conducted signals masked by air conducted noise, the individual differences in thresholds are controlled by the variations between listeners in both the bone-conduction and air-conduction pathways

The results on the variability of bone conduction thresholds presented here are in good agreement with the results of Studebaker (1962 *b*) and those summarized by him

# VI. SUMMARY AND CONCLUSIONS

The primary purpose of this study was to develop and evaluate a method for obtaining monaural thresholds for bone-conducted signals (tones) masked by air conducted noises The method required air-conducted noise (masking noise) at the ear at which bone conduction thresholds were determined and another air-conducted noise (blocking noise) at the other ear The blocking noise was used to eliminate functionally one ear from the experiment The evaluation of this method indicated that the blocking noise must be both independent of and about 25 dB higher in level than the masking noise These conclusions were supported by Experiment I for the case of bone conducted signals and air-conducted noises and by Experiment II for the case of signals and noises by air conduction Both of these experiments in turn supported the results of Weston and Miller (1965)

Experiment III provided several kinds of information

*Masking functions* The shapes of the curves relating monaural masked threshold to noise level were identical (linear with slope one after an initial curvilinear section) for air- and bone-conducted signals except when both the Pedersen Receivers (large volume) were over the ears and the bone-conducted signal was 250 or 500 cps The deviant masking functions are probably due to the action of the intra-aural muscles This seems reasonable since, (1) blocking noise at these levels produced a change in the acoustic impedance at the eardrum and thus contraction of the intra-aural muscles can be inferred, and (2) when the Pedersen Receivers were over the ears, the bone-conduction and air-conduction pathways were probably differentially affected by the action of the intra-aural muscles

*The occlusion effect* The "occlusion effect" is maintained at masked as well as quiet threshold The magnitudes of the occlusion effect for the Conventional Receivers (TDH-39 earphones mounted in MX-41/AR cushions) compared to the Pedersen Receivers (large volume) were about 22, 18, 10, and $\pm 3$ dB for frequencies of 250, 500, 1000, and 2000 cps, respectively

*Physical measures of the threshold signals* Physical measures were calculated for both air- and bone conducted signals at threshold intensity For air-conducted signals they were calculated in terms of coupler-calibration measurements, while for bone-conducted signals a velocity-sensitive measurement system attached to the bone-conduction transducer was used The quiet thresholds for both air- and bone conducted signals were in good agreement with similar data reported in the literature (see Figs 5 and 6)

*Possible applications of methods and results* The amount of air conducted noise required to produce a specific amount of masking of a bone conducted signal at an ear can be determined from the data These determinations can be made for two limiting cases that of a sizable occlusion effect (*TDH 39* earphone mounted in a MX-41 AR cushion) and that of no occlusion effect (Pedersen Receivers) Determinations for other earphone cushion volumes can be interpolated

The fact that most of the masking functions were found to be linear with *slope one means that the method under certain conditions provides a means* for the calibration of bone conduction transducers at input levels greater than those required at quiet threshold

*Variability of thresholds* The variability of masked thresholds within listeners conditions and replications was small for all experimental conditions The variability within listeners and conditions but over replications was larger than the variability within listeners conditions and replications and this increase in variability was greater for bone than for air conducted signals Individual differences were larger than either kind of within listener variability For air conducted signals individual differences were smaller for masked thresholds than for quiet thresholds while for bone conducted signals individual differences for both quiet thresholds and those masked by air conducted noise were nearly the same Individual differences in thresholds were much larger for bone than for air conducted signals

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# APPENDIX A

## Apparatus

The apparatus used in these experiments will be described under five headings First, the overall properties of the channels used for noises and tones will be described and then the transducers and the apparatus used for their control and calibration will be described in detail

*Noise and tone channels*

The essential features of the noise and tone channels are shown in Fig 7 There are three channels for generating tones and noises one for the right earphone (right channel), one for the left earphone (left channel), and one for the bone-conduction transducer (bone channel)

Consider now the right and left channels In each of these channels the output of a noise generator (CID No 24 or Grason Stadler, Model No 455B) was led to a low pass filter (Spencer Kennedy Labs, Model No 302) The low pass filters had a rejection rate of 18 dB per octave above the cut-off frequencies which were set to either 700 or 7000 cps The output of the filter in each channel was then amplified (Langevin Model 117 A in the right channel and General Radio, Type 1206 B in the left) The amplifier outputs could be switched by Switch No 1 so that the initial portion of the left channel was disconnected and the output of the amplifier in the right channel was led into the remainder of both the left and right channels In another position of Switch No 1 the right and left channels remained isolated, each being connected to its own noise generator, filter and amplifier combination Two remaining positions of Switch No 1 allowed noise to be introduced into only one of the two channels either the right or left The levels at the outputs of the switch were maintained constant independent of the switch position by means of fixed loss pads within the switch circuitry The vertical and horizontal inputs of an oscilloscope (Heathkit Model IO 10) were bridged across the outputs of Switch No 1 in order to provide a visual monitor of both noise channels and their correlation

Each output of Switch No 1 was fed to a set of precision decade attenuators (Langevin Models AT 505 and AT 510) and each set had a maximum attenuation of 110 dB These attenuators were variable in 1 dB steps The output of the attenuator set in each channel fed a three way resistive mixing network The output of each mixer was fed in turn to an impedance

Fig 7 Block diagram of the electronic apparatus

matching transformer (UTC, Model CVL-1) The transformers were strapped for an input impedance of 600 ohms and an output impedance of 10 ohms

A two position switch ($SW_4$) was bridged across the outputs of the transformers and connected to an AC voltmeter (Ballantine, Model 300) for voltage calibrations of each channel Cables connected the outputs of the transformers to a terminal board in the quiet room for connection to the earphones

Pure tones could be generated and mixed into the right, left, or bone channels by the following series of devices The output of a pure-tone oscillator (Hewlett-Packard, Model 241A) was fed to an electronic switch (CID No 53), triggered by means of a square wave generator (CID No 53), so that a pulsed tone (on-off ratio, 50%, period, 500 msec, and rise-fall time, 20 msec) was available as a signal This signal was fed to two precision attenuators in tandem The first was an attenuator set (Langevin, Models AT-505 and AT-510) with a maximum attenuation of 110 dB in steps of 1 dB and this attenuator was controlled by the experimenter The second attenuator (Daven, Model Spec 4388G) had a maximum attenuation of 45 dB with 1-dB steps, it was under the control of the subject and located in the quiet room A 10-dB pad could be inserted at the output of this attenuator by means of a switch located in the same box as the attenuator

The output of the subject's attenuator was connected to a three-position switch, Switch No 3 ($SW_3$) By means of this switch the pulsed tone could be introduced into the bone channel, or, for measurements of thresholds by air conduction, was connected to the inputs of two identical line amplifiers (CID No 141) whose outputs each fed one of the three way mixing networks in the right and left channels The line amplifiers were used to pro-

vide better than 80-dB isolation between the two channels Switch No 2 (SW.) allowed the pulsed tone to be fed to either the right or the left channels or to both channels simultaneously

When Switch No 3 was set so that the pulsed tone was to be fed into the bone channel the circuit was as follows The output of the subject s attenuator was fed to a fixed attenuator (not shown) and then to a power amplifier (McIntosh Model 50W 2) whose output was terminated at the bone-conduction transducer A VU meter (Daven Model 910 E) was bridged across the output of the power amplifier for voltage calibrations

In order to facilitate both the rapid and accurate reading of the subject s and experimenter s attenuators these attenuators were modified by the addition of rotary switches so that their settings were visually displayed at the experimenter s control desk by means of one plane digital display units (Industrial Electronic Engineers Model No *10010*)

Several important characteristics of the three channels are described below The frequency responses of the right and left noise channels with the low pass filters switched to the 7000-cps cut off were measured at the terminal board in the quiet room The response for each channel was flat (+0 2 dB) from 100 to 5000 cps and 3 dB down at the cut off frequency The rejection rate beyond cut off was 18 dB per octave The right left and *bone channels had a frequency response that was flat (+0 5 dB) from 100* to 10 000 cps The electrical phase shift between the right and left channels was less than 2 degrees at 500 cps and about 3 6 degrees at 5000 cps Measurements of harmonic distortion in the portion of the right and left channels used for the pulsed tones indicated that the second harmonic was about 35 to 40 dB below the fundamental over the frequency range that was used while the higher harmonics were down 50 dB or more Attenuator linearity was maintained at least to below 100 dB below 0 1 volt across the earphone terminals At this level the electrical noise masked further measurements

*Earphones*

The two types of earphones or receivers used have been described previously (see Chapter II) They were designated Conventional Receivers and Pedersen Receivers

Sound pressure calibrations were performed for both sets of receivers by both continuous and discrete frequency methods The additional instrumentation used for these calibrations was a sweep frequency oscillator amplifier and level recorder (Bruel & Kjaer (B & K) Audio Frequency Response and Spectrum Recorder Model 3326) laboratory standard microphone B & K Model 4132) and associated cathode follower (B & K Model 2619 Two types of acoustic couplers (artificial ears) were used for these *calibrations An ASA* Type 1 coupler (B & K Model DB 0161) was used for the Conventional Receivers while an NBS 9A coupler B & K Model DB 0160 with an extended plate was used for the Pedersen Receivers The

TABLE IX *Sound pressure levels (SPL's) in decibels re 0.0002 μbar for the two types of receivers at the four experimental frequencies*

Source 0.1 volt

| Frequency (cps) | Conventional Receivers | | Pedersen Receivers | |
|---|---|---|---|---|
| | Right | Left | Right | Left |
| 250 | 107.2 | 106.6 | 98.6 | 102.4 |
| 500 | 107.2 | 106.3 | 100.4 | 101.4 |
| 1000 | 106.4 | 105.3 | 93.7 | 93.7 |
| 2000 | 104.3 | 104.9 | 85.8 | 86.4 |

NBS-9A coupler was extended by means of an additional plate in order to provide a hard, flat surface against which the Pedersen Receivers could rest for a good and reproducible acoustic seal (Davis, Cox and Glorig, 1963; Shaw and Thiessen, 1962).

All pressure calibrations were performed with 0.1 volt across the receiver terminals Calibrations were done both prior to and at the completion of each experiment No changes in the calibrations of either of the sets of receivers were observed except in Experiment I The calibrations in sound-pressure levels at the four experimental frequencies used in the experiments are shown for both sets of receivers in Table IX

### *Bone-conduction transducer*

A commercially available dynamic-type bone-conduction transducer was used (Mrico, Model C) The moving-iron piston, or drive rod, of the transducer was extended in length by means of a plastic rod and terminated with a 1.5 cm² chamfered plastic tip The cylindrical body of the transducer was fitted to a rubber sleeve which in turn was mounted in a housing of brass The brass housing and the drive rod terminated with the plastic tip are shown in Fig 8, and in more detail, in Fig 11

Figure 8 also shows the apparatus used to mount rigidly the transducer housing and assembly The transducer could be extended to the required point of application on the head by means of a rack and pinion connected to a vernier adjustment knob Both elevation and azimuth adjustments of the transducer could be made by sliding the transducer carriage along the steel semi-circular track and by rotation of the track around its vertical axis, respectively The steel track in turn was mounted on a two-by-eight wooden beam which was suspended between two of the walls in the quiet room Only adjustments of elevation were made in the experiments reported, [illegible] were required because of the variations in frontal-bone contours



Fig 8 Apparatus for mounting the bone conduction transducer and pickup system

among the subjects The subject was seated in a dental chair and, therefore, could easily be raised to the correct position relative to the transducer for application and positioning of the transducer on the head

*Calibration and monitoring of the bone-conduction transducer*

Two measurement systems were used for the calibration of the bone conduction transducer One of the systems was also used to monitor the dynamic behavior of the transducer during the experiments Measurements of the response of the transducer were obtained in terms of rms velocity

One measurement system was an accelerometer (B & K, Model 4328) and preamplifier-equalizer complement (B & K, Model 1606) The system could be used to obtain vibration measurements in terms of acceleration, velocity, or displacement This system was highly accurate but could not be used to measure the velocity of the bone-conduction transducer when the transducer was coupled to a human head However, this accelerometer system could be used to calibrate the bone conduction transducer when the transducer was coupled to an "artificial" load

The other measurement system was a ceramic phonograph cartridge (Astatic, Model 51-1) and a preamplifier-equalizer complement which was specially constructed for this system This system was designed for vibration measurements of the bone-conduction transducer when it was, in fact,

coupled to the head This system is called the "pickup" system Because the pickup system was the primary means of calibrating and monitoring the response of the bone-conduction transducer, it is described in detail below

When a ceramic phonograph pickup is subjected to lateral motion (from its needle tracking the modulated grooves of a phonograph record for example), it produces an open-circuit voltage which is essentially proportional to the amplitude of this motion In the present case, the pickup was attached to the housing of the bone conduction transducer in such a way that its needle was forced to follow the rectilinear motion of the transducer's drive rod The output voltage of the pickup was then proportional to the amplitude of the vibrational motion of the bone conduction transducer

However, the rectilinear motion of the bone-conduction transducer, because of the dynamic characteristics of such devices, is more conveniently described in terms of velocity than in terms of amplitude Therefore, it was decided to equalize the electrical output of the pickup so that its response could also be described in terms of velocity

The electronic device used to equalize the pickup response consisted of four components a cathode follower, two identical equalization stages, and a line amplifier The cathode follower was used to match the impedance of the pickup to that of the first stage of variable equalization The two equalization stages were connected in tandem Each stage could alter the frequency response at pre determined points in the frequency spectrum (Barhydt, 1956) The output of the second stage was connected to a line amplifier with an output impedance of 600 ohms

Now in order to obtain the necessary equalization of the pickup, a calibrated source of vibratory signals was required to drive the pickup Two such calibrated sources were available in the form of phonograph records One record (CBS Labs, Type STR-100) contained a sweep frequency band from 20 to 20,000 cps recorded at specified velocity levels The other record (RCA, Type 12-5-50) was similar to the one above except it contained discrete frequencies Both records were recorded with the following frequency characteristics constant amplitude for frequencies up to 500 cps and constant velocity for frequencies beyond 500 cps

The output of an ideal velocity-sensitive pickup when reproducing the frequency characteristics of the two records would be as follows an output level increasing with frequency at the rate of 6 dB per octave for frequencies below 500 cps, and a constant output level for frequencies above 500 cps This ideal response is illustrated by the dashed line in Fig 9 The solid line, on the other hand, shows the response of the pickup system and it is the mean of the responses obtained from the two records The agreement between the frequency-response characteristics obtained using the two re-ls was excellent ($\pm 1$ dB between 200 and 6000 cps) Since the experi-n limited to test frequencies between 200 and 4000 cps the devia-

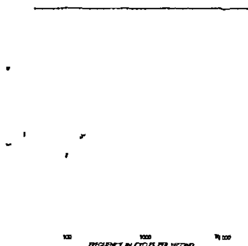



Fig 9 The mean response of the velocity sensitive pickup system obtained from two calibration records as sources The ideal response is shown by the dashed lines while the solid line shows the actual response

tions of the measured curve from the theoretical curve shown in Fig 9 are of no concern

The relations between the output voltages of the pickup system and the velocities on the records were obtained from measurement of the voltages produced by the recorded velocities These recorded velocities were specified by the manufacturers of the records The entire pickup system was adjusted so that an output of 1 0 volt was obtained for an input velocity of 1 0 cm/sec

It was assumed that the calibration of the pickup system as described above would be maintained when the pickup itself was connected to the drive rod of the bone conduction transducer Therefore in order to test this assumption comparison calibrations were obtained for the bone conduction transducer by means of the pickup and the accelerometer systems

The comparison calibrations of the bone-conduction transducer were obtained with an artificial load coupled to the transducer s drive rod As previously mentioned this was necessary because the accelerometer system could not be used when a human head provided the load With the pickup and accelerometer attached to the transducer a 500 gram cylindrical mass was mounted concentrically on the tip of the drive rod The comparison calibrations were performed with the transducer in the position as shown in Fig 12

In general the results of these calibrations between the pickup and the accelerometer systems were in good agreement between 200 and 4000 cps However the calibration response obtained by the pickup system exhibited minor resonant peaks at several frequencies above 800 cps These resonant

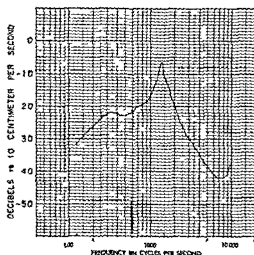



Fig 10 Calibration curve for the bone-conduction transducer obtained by means of the pickup system The transducer was in place on the frontal bone and a coupling force of 750 grams was used

peaks were attributed to the artificial load, because they were not observed when the head was used as a load Assuming that these minor resonances can then be neglected in this case, the difference between the calibrations obtained by the two systems for the transducer with the artificial load was ± 1 dB from 200 to 4000 cps

Therefore, it was concluded that the pickup system was accurately calibrated (± 1 dB) over the frequency range from 250 to 2000 cps which was the range used in the experiments

A calibration curve for the bone-conduction transducer coupled to a human head was obtained by means of the pickup system For these measurements the transducer was in place on the frontal bone and a coupling force of 750 grams was used The results are shown in Fig 10 It should be noted that the calibration curve indicates that the fundamental resonant frequency of the bone-conduction transducer is about 1400 cps In contrast, the resonant frequency obtained for the calibrations done with the artificial load was about 800 cps The upward frequency shift of the resonant peak when the head was used as the load on the transducer is probably due to a basic characteristic feature of the transducer This characteristic feature is that the vibratory response of the transducer is not independent of the applied load However, with a coupling force of 750 grams and the head as a load the stability of the response of the transducer was good Only minor variations in the response curve shown in Fig 10 were observed from head to head

As a further precaution against the possibility that the calibration of the bone conduction transducer might change during the actual experiments, [illegible] calibration measurements were performed prior to and at the com-

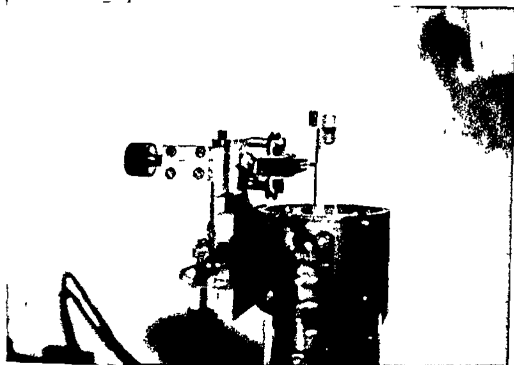


Fig 11 Mechanical arrangement used to mount the pickup on the bone conduction transducer The needle of the pickup can be seen mounted in the brass stud on the drive rod

pletion of each experimental session during which bone-conduction thresholds were determined Therefore, any shift in the resonant frequency (and, therefore a shift of the response curve as a whole) would be included in these calibration measurements

The manner in which the pickup was mounted on the housing of the bone-conduction transducer and the location of the pickup relative to the drive rod of the transducer are shown in Fig 11 The pickup was mounted so that the needle was perpendicular to the drive rod The needle was fitted into a small brass stud which in turn was mounted on the drive rod near its tip The mounting arrangement provided for movement of the pickup in three dimensions (parallel, perpendicular, and lateral) relative to the drive rod This was necessary in order to align the needle accurately with the stud into which it must fit The pickup was mechanically isolated from its adjustable mounting arrangement by means of rubber washers

*System for maintaining the coupling force*

The system used to maintain the coupling force between the bone-conduction transducer and the frontal bone of the subject's head is shown in Fig 12 This system and one which was investigated for possible use are described below

# APPENDIX B

## Table of Mean Variances

## Table of Standard Deviations

TABLE X

| Frequency (cps) | Bone conduction | | | | Air conduction | | | |
|---|---|---|---|---|---|---|---|---|
| | Conventional | | Pedersen | | Conventional | | Pedersen | |
| | Quiet | Masked | Quiet | Masked | Quiet | Masked | Quiet | Masked |
| *Mean variances[a] for measures within listeners conditions and replications* ($\sigma^2_{W_{LCR}}$) | | | | | | | | |
| 250 | — | 1.65 | — | 2.54 | — | 1.32 | — | 1.25 |
| 500 | — | 1.43 | — | 1.72 | — | 1.28 | — | 1.96 |
| 1000 | — | 1.56 | — | 1.41 | — | 1.36 | — | 1.99 |
| 2000 | — | 1.73 | — | 1.20 | — | 1.75 | — | 0.98 |
| Mean $\sigma^2$ | — | 1.489 | — | 1.723 | — | 1.427 | — | 1.541 |
| Mean[b] $\sigma_x^2$ | — | 0.745 | — | 0.862 | — | 0.714 | — | 0.777 |
| *Mean variances[a] for measures within listeners and conditions but between replications* ($\sigma^2_{W_{LC}B_R}$) | | | | | | | | |
| 250 | 7.150 | 7.562 | 3.925 | 2.112 | 3.000 | 1.317 | 1.625 | 1.512 |
| 500 | 7.425 | 5.478 | 8.775 | 5.949 | 5.150 | 1.756 | 1.200 | 1.816 |
| 1000 | 6.700 | 8.361 | 13.325 | 9.609 | 2.650 | 1.391 | 3.950 | 1.399 |
| 2000 | 7.350 | 4.248 | 3.300 | 3.058 | 3.975 | 2.500 | 3.300 | 1.191 |
| Mean $\sigma^2$ | 7.156 | 6.413 | 7.331 | 5.182 | 3.694 | 1.749 | 2.519 | 1.479 |
| *Mean variances[a] for measures within conditions and replications but between listeners* ($\sigma^2_{W_{CR}B_L}$) | | | | | | | | |
| 250 | 10.563 | 22.750 | 12.886 | 9.975 | 6.296 | 4.420 | 4.820 | 3.860 |
| 500 | 6.636 | 13.052 | 39.766 | 21.721 | 4.946 | 1.673 | 5.886 | 3.618 |
| 1000 | 56.330 | 31.322 | 59.933 | 44.698 | 7.020 | 1.900 | 12.696 | 1.870 |
| 2000 | 20.996 | 82.676 | 17.786 | 42.795 | 23.580 | 2.352 | 19.813 | 2.247 |
| Mean $\sigma^2$ | 23.624 | 38.200 | 32.592 | 29.795 | 10.460 | 2.586 | 10.800 | 2.898 |

[a] Mean $\sigma^2$ is the mean of the mean variances shown for each frequency

[b] For explanation see Chapter V section entitled "Variability of Thresholds"

TABLE XI

| Frequency (cps) | Bone conduction | | | | Air conduction | | | |
|---|---|---|---|---|---|---|---|---|
| | Conventional | | Pedersen | | Conventional | | Pedersen | |
| | Quiet | Masked | Quiet | Masked | Quiet | Masked | Quiet | Masked |
| *Standard deviations[a] for measures within listeners conditions and replications* ($\sigma_{W_{LCR}}$) | | | | | | | | |
| 250 | — | 1 285 | — | 1 591 | — | 1 149 | — | 1 118 |
| 500 | — | 1 196 | — | 1 311 | — | 1 131 | — | 1 100 |
| 1000 | — | 1 219 | — | 1 200 | — | 1 166 | — | 1 411 |
| 2000 | — | 1 315 | — | 1 095 | — | 1 323 | — | 0 989 |
| Mean $\sigma$ | — | 1 220 | — | 1 313 | — | 1 195 | — | 1 242 |
| Mean $\sigma_{\bar{x}}$ | — | 0 863 | — | 0 928 | — | 0 845 | — | 0 881 |
| *Standard deviations[a] for measures within listeners and conditions but between replications* ($\sigma_{W_{LC}B_R}$) | | | | | | | | |
| 250 | 2 674 | 2 749 | 1 981 | 1 453 | 1 732 | 1 161 | 1 275 | 1 229 |
| 500 | 2 725 | 2.341 | 2 962 | 2 439 | 2 269 | 1 325 | 1 095 | 1 347 |
| 1000 | 2 588 | 2 892 | 3 650 | 3 099 | 1 628 | 1 181 | 1 987 | 1 183 |
| 2000 | 2 711 | 2 061 | 1 817 | 1 749 | 1 994 | 1 581 | 1 817 | 1 091 |
| Mean $\sigma$ | 2 675 | 2 532 | 2 708 | 2 276 | 1 922 | 1 323 | 1 587 | 1 216 |
| *Standard deviations[a] for measures within conditions and replications but between listeners* ($\sigma_{W_{CR}B_L}$) | | | | | | | | |
| 250 | 3 246 | 4 769 | 3 589 | 3 158 | 2 509 | 2 102 | 2 195 | 1 965 |
| 500 | 2 576 | 3 613 | 6 306 | 4 661 | 2 924 | 1 292 | 2 426 | 1 902 |
| 1000 | 7 505 | 5 858 | 7 742 | 6 686 | 2 649 | 1 378 | 3 513 | 1 367 |
| 2000 | 4 582 | 9 093 | 4 217 | 6 542 | 4 856 | 1 534 | 4 451 | 1 499 |
| Mean $\sigma$ | 4 860 | 6 181 | 5 709 | 5 459 | 3 934 | 1 608 | 3 286 | 1 702 |

[a] All entries are square roots of the corresponding numbers in Table X

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# THEORETICAL ASPECTS OF THE ROLE OF ANGULAR ACCELERATION IN VESTIBULAR STIMULATION

BY

R. S. WEAVER

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM O

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SUPPLEMENTUM 205

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## CONTENTS

# INTRODUCTION

Studies in vestibular physiology are currently of major importance in the areas of space research and high speed flight Experimental facilities such as human centrifuges with freely rotating gondolas are becoming more common the forces imposed on humans in this equipment and in flight are becoming greater and increasingly complex A knowledge of both direction and magnitude of these imposed forces is essential for the physiologist to fully understand experimental results and specify human tolerances

Most complex physical effects are combinations of simpler phenomena in rotational motion for example a complex acceleration may be composed of a combination of linear and angular accelerations This treatise systematically considers different accelerations and shows that turntable accelerations generally consist of several components each readily calculable

The first part of the main section applies the general velocity equation to particular types of rotation with special reference to turntable experiments The second part is concerned with the accelerations experienced by these moving subjects again illustrated by experiments involving rotation on a turntable For differing conditions of rotation the acceleration equation is applied to the inner ear and directions and magnitudes of resultant stimuli are calculated and compared with physical sensations Finally an example of a problem arising in a centrifuge experiment is included

The use of vector notation rather than trigonometric functions throughout the report leads to simplified expressions as well as easier calculation of magnitudes and visualization of results In Appendix I a concise introduction to vector algebra and vector notation used in this work is developed This introductory appendix is intended for those with little or no experience in vectors The general equations of motion are developed in vector form in Appendix II Appendix III consists of graphs from which the resultant acceleration in simple rotation can be determined from a knowledge of the parameters of the rotation Appendix IV deals with the anatomy of the inner ear For those unfamiliar with vector mathematics and those wishing to become more familiar with the notation employed here this report should be read starting at Appendix I For readers already acquainted with vector mathematics a brief summary of vector operations needed and notation used in the main body of the report follows

# APPLICATIONS OF EQUATIONS OF MOTION

The vector equations of motion can be used to determine the direction and magnitude of the velocity and acceleration of any point in a moving system. In the following sections these equations are applied to situations in which a human subject is part of a rotating system, including conditions of head movement which are known to stimulate the vestibular apparatus.

In the following sections, the origin of the fixed or inertial coordinate system normally coincides with the centre of the turntable, the origin of the moving coordinate system is normally fixed in the chair of the subject on the turntable. There are then three kinds of rotation:

$\Omega$ = angular velocity of the turntable

$\omega$ angular velocity of the moving axes system with respect to the fixed axes system (if the chair is fixed to the turntable $\Omega = \omega$) and

W angular velocity of the subject with respect to the moving axes system (his chair)

## VELOCITY

$$\dot{\mathbf{r}}_0 = \dot{\mathbf{R}} + \dot{\mathbf{r}}$$
$$= \dot{\mathbf{R}} + \mathbf{v} + \boldsymbol{\omega} \times \mathbf{r}$$

where $\mathbf{v}$ is the velocity of a point with respect to the moving axes and $\boldsymbol{\omega}$ is the *angular* velocity of the moving axes with respect to the inertial axes.

### 1 Basic Conditions

A seated subject is rotated on a turntable and, while free to move, faces the centre of rotation at all times.

In this case the subject (or the moving coordinate system of which he may be considered a part) has the same angular velocity $\boldsymbol{\omega}$ as the angular velocity $\boldsymbol{\Omega}$ of the turntable (with respect to the fixed (inertial) coordinate system).

Now write $\qquad \mathbf{R} = R_x\mathbf{i}_0 + R_y\mathbf{j}_0 + R_z\mathbf{k}_0$

where $\mathbf{i}_0$, $\mathbf{j}_0$ and $\mathbf{k}_0$ are *unit* vectors (vectors of unit length) in the inertial system.

Then $\qquad \dot{\mathbf{R}} = \dot{R}_x\mathbf{i}_0 + \dot{R}_y\mathbf{j}_0 + \dot{R}_z\mathbf{k}_0 + R_x\dot{\mathbf{i}}_0 + R_y\dot{\mathbf{j}}_0 + R_z\dot{\mathbf{k}}_0$

For the inertial frame the unit vectors do not change with time, hence $\dot{\mathbf{i}}_0 = \dot{\mathbf{j}}_0 = \dot{\mathbf{k}}_0 = 0$. Since $\mathbf{R}$ is the radius of a circle, then $\dot{\mathbf{R}}$ is the velocity of a point on the circle and therefore $\dot{\mathbf{R}} = \boldsymbol{\Omega} \times \mathbf{R}$.

# VECTOR NOTATION

*Scalars* are observable quantities having only a magnitude, such as *temperature*, and may be written as $T$. *Vectors* are used to represent quantities having both a magnitude and an associated direction, such as *force*; they may be written as $\mathbf{F}$, where $F$ is the magnitude of the vector $\mathbf{F}$. Vectors are normally shown graphically as arrows whose length represents the magnitude of $\mathbf{F}$ and whose direction is along the direction of action of $\mathbf{F}$. Addition and subtraction of vectors is easily described and shown graphically (for example, see Figure 14, page 25). Two kinds of vector multiplication are defined. The *inner* or *dot product* of $\mathbf{a}$ and $\mathbf{b}$ is $\mathbf{a} \cdot \mathbf{b}$; this leads to a scalar of magnitude $ab \cos \theta$, where $\theta$ is the smaller angle between the vectors $\mathbf{a}$ and $\mathbf{b}$. The *vector* or *cross product* of two vectors, written as $\mathbf{a} \times \mathbf{b}$, is defined as another vector with magnitude $ab \sin \theta$ and direction at right angles to the plane containing $\mathbf{a}$ and $\mathbf{b}$. (The vector $\mathbf{a} \times \mathbf{b}$ points in the direction of advance of a right handed screw turning from $\mathbf{a}$ to $\mathbf{b}$.) *Angular* velocities and accelerations can both be represented vectorially as follows. The angular vector points along the axis of rotation and is again directed by the right hand screw rule; the magnitude of the angular vector represents the magnitude of the angular quantity (either angular velocity or angular acceleration). A list of the symbols used in this report and page references to them is summarized in Table 1, page 37.

For calculating the accelerations or forces experienced at any point in a subject, two Cartesian coordinate systems will be used: an inertial system, such as a set of axes fixed to the earth, and a moving set of axes normally fixed in the subject or his chair. The *displacement* or position vector of a point in the moving system is shown in Figure 17, page 30. Here $\mathbf{r}_0$ is the displacement of the point with respect to the fixed or inertial axes; $\mathbf{r}_0$ is the vector sum of two displacements $\mathbf{R}$ and $\mathbf{r}$. $\mathbf{R}$ is the displacement of the origin of the moving axes with respect to the inertial frame, and $\mathbf{r}$ is the position of a point in the moving system with respect to the origin of this system. Equations for velocity and acceleration of this point are obtained by

(i) finding the rate of change with time of the displacement to obtain *velocity*, for example $\mathbf{v} = \dot{\mathbf{r}}$;

(ii) finding the rate of change with time of the velocity to obtain *acceleration*, for example $\mathbf{a} = \dot{\mathbf{v}} = \ddot{\mathbf{r}}$.

A dot over a quantity conventionally indicates a time rate of change, hence $\dot{\mathbf{r}} = \mathbf{v}$ and $\ddot{\mathbf{r}} = \dot{\mathbf{v}} = \mathbf{a}$. It will be noted that these time rates of change include both the magnitude and the direction of the vector. The derivations of the general motion equations and a complete description of the notation are outlined in Appendix II.

## APPLICATIONS OF EQUATIONS OF MOTION

The vector equations of motion can be used to determine the direction and magnitude of the velocity and acceleration of any point in a moving system In the following sections these equations are applied to situations in which a human subject is part of a rotating system including conditions of head movement which are known to stimulate the vestibular apparatus

In the following sections the origin of the fixed or inertial coordinate system normally coincides with the centre of the turntable the origin of the moving coordinate system is normally fixed in the chair of the subject on the turntable There are then three kinds of rotation

- $\Omega$ angular velocity of the turntable
- $\omega$ angular velocity of the moving axes system with respect to the fixed axes system (if the chair is fixed to the turntable $\Omega = \omega$) and
- $W$ angular velocity of the subject with respect to the moving axes system (his chair)

## VELOCITY

$$\mathbf{r}_0 = \mathbf{R} + \mathbf{r}$$

$$\dot{\mathbf{R}} + \mathbf{v} \quad \omega \times \mathbf{r}$$

where v is the velocity of a point with respect to the moving axes and $\omega$ the *angular* velocity of the moving axes with respect to the inertial axes

### 1 Basic Conditions

A seated subject is rotated on a turntable and while free to m the centre of rotation at all times

In

be co

$\Omega$ of ntre

is $\Omega$

Now write $\mathbf{R} = R_x\mathbf{i}_0 + R_y\mathbf{j}_0 + R_z\mathbf{k}_0$ the

where $\mathbf{i}_0$ $\mathbf{j}_0$ and $\mathbf{k}_0$ are *unit* vectors (vectors of unit leng system

Then $\dot{\mathbf{R}} \quad \dot{R}_x\mathbf{i}_0 + \dot{R}_y\mathbf{j}_0 + \dot{R}_z\mathbf{k}_0 + R_x\dot{\mathbf{i}}_0 + R_y\dot{\mathbf{j}}_0 + R$ he tem

For the inertial frame the unit vectors do not c ration $\dot{\mathbf{i}}_0 \quad \dot{\mathbf{j}}_0 = \dot{\mathbf{k}}_0 = 0$ Since R is the radius of a circle thr itude for point on the circle and therefore $\dot{\mathbf{R}} = \Omega \times \mathbf{R}$ ates

Then
$$\mathbf{r}_0 = \mathbf{R} + \mathbf{v} + \boldsymbol{\omega} \times \mathbf{r}$$
$$= \boldsymbol{\Omega} \times \mathbf{R} + \mathbf{v} + \boldsymbol{\omega} \times \mathbf{r}$$
$$= \boldsymbol{\omega} \times (\mathbf{R} + \mathbf{r}) + \mathbf{v} \text{ since } \boldsymbol{\omega} = \boldsymbol{\Omega}$$
$$= \boldsymbol{\omega} \times \mathbf{r}_0 + \mathbf{v}$$

Hence the linear velocity of any part of this subject is the vector sum of the velocity $\boldsymbol{\omega} \times \mathbf{r}_0$ due to turntable rotation and the velocity $\mathbf{v}$ of the part with respect to the origin of the moving system

*Subcondition (1a)*

Subject is fixed in his chair

Then for all parts of his body $\mathbf{v} = 0$ and the velocity of any part becomes $\mathbf{r}_0 = \boldsymbol{\omega} \times \mathbf{r}_0$ Here the velocity of any point in the body depends solely on the rate of rotation of the turntable and on the distance of the point from the centre of the turntable

## 2 Basic Conditions

Subject is seated on a rotating turntable and is free to move but always faces the same direction Then he has translational motion but no rotation with respect to the inertial coordinate frame

Thus $\boldsymbol{\omega} = 0$ and the velocity becomes
$$\mathbf{r}_0 = \boldsymbol{\Omega} \times \mathbf{R} + \mathbf{v}$$

*Subcondition (2a)*

Subject is fixed in his chair

Then $\mathbf{v} = 0$ and $\mathbf{r}_0 = \boldsymbol{\Omega} \times \mathbf{R}$

In this case the velocities of all points in the body depend only on the rate of rotation of the turntable and the distance from the centre of the turntable to the centre of the moving coordinate system (that is the pivot of his chair) Therefore all points in the subject move with equal velocities regardless of their distance from the centre of the turntable This means that all parts of the subject move with the same angular velocity in circles of the same radius but the axes of these circles do not necessarily coincide with one another or with the axis of the turntable

## 3 Basic Conditions

The subject is seated on a rotating turntable in a chair which rotates with respect to the rotating turntable Any point in the subject at a given instant is at a distance $r$ from the centre 0 of the *moving* axes and is rotating with angular velocity $\mathbf{W}$ around 0

Then the velocity of the point with respect to the moving axes system is
$$\mathbf{v} = \mathbf{W} \times \mathbf{r}$$

Hence
$$\mathbf{r}_0 = \mathbf{R} + \mathbf{v} + \boldsymbol{\omega} \times \mathbf{r}$$
$$= \boldsymbol{\Omega} \times \mathbf{R} + \mathbf{W} \times \mathbf{r} + \boldsymbol{\omega} \times \mathbf{r}$$
$$= \boldsymbol{\Omega} \times \mathbf{R} + (\mathbf{W} + \boldsymbol{\omega}) \times \mathbf{r}$$

$\mathbf{r}$ will be a function of both $\mathbf{W}$ and time under these conditions

*For example if a point in the moving system rotates in a circle about the* $Z$ axis then $\mathbf{W} = W\mathbf{k}$ since only a $Z$ component of angular velocity exists The path traced out by this point may be represented by the equation $\mathbf{r} = (r \cos Wt)\mathbf{i} + (r \sin Wt)\mathbf{j} + \mathbf{k}$ The velocity of the point is $\mathbf{v} = \mathbf{W} \times \mathbf{r} = -(Wr \sin Wt)\mathbf{i} + (Wr \cos Wt)\mathbf{j}$

This proves that as the point rotates around the $Z$ axis its velocity in a particular direction (such as the $X$ direction) will vary periodically from $+Wr$ to zero to $-Wr$ to zero to $+Wr$ Since Coriolis acceleration is a function of velocity the magnitude of the Coriolis acceleration at a point in a semicircular canal may change *as the head rotates even when both the head* and the turntable rotate at constant angular velocities

## ACCELERATION

$$\mathbf{r}_0 = \mathbf{R} + \mathbf{a} + 2\boldsymbol{\omega} \times \mathbf{v} + \boldsymbol{\alpha} \times \mathbf{r} + \boldsymbol{\omega} \times (\boldsymbol{\omega} \times \mathbf{r})$$

Here $\mathbf{a}$ is the linear acceleration of a point with respect to the moving co ordinate system and $\boldsymbol{\alpha}$ is the angular acceleration of the moving axes with respect to the inertial axes (see Appendix II page 29)

### 1 Basic Conditions

In a turntable experiment the subject is fixed in his chair (that is he is at rest with respect to the moving axes system The system however is free to move in any manner)

Then $\mathbf{a}$ and $\mathbf{v}$ are both zero and

$$\mathbf{r}_0 = \mathbf{R} + \boldsymbol{\alpha} \times \mathbf{r} + \boldsymbol{\omega} \times (\boldsymbol{\omega} \times \mathbf{r})$$

*Subcondition (1 a)*

In many experiments the distance of the subject s chair from the centre of the turntable is fixed and the turntable has a constant angular velocity $\boldsymbol{\Omega}$

Then $\mathbf{R} = \boldsymbol{\Omega} \times (\boldsymbol{\Omega} \times \mathbf{R})$ that is this centrifugal acceleration acts on the subject due to rotation of the turntable

*Subcondition (1 b)*

If in addition the subject faces always in the same direction so that he is translating but not rotating with respect to the inertial coordinate system then $\boldsymbol{\alpha}$ and $\boldsymbol{\omega} = 0$ and $\mathbf{r}_0 = \boldsymbol{\Omega} \times (\boldsymbol{\Omega} \times \mathbf{R})$ In this case the only acceleration experienced by the subject is a centrifugal one constant in magnitude for all parts of the subject but varying in direction as the turntable rotates

Then $$r_0 = R + v + \omega \times r$$
$$\Omega \times R + v + \omega \times r$$
$$\omega \times (R + r) + v \quad \text{since } \omega = \Omega$$
$$\omega \times r_0 + v$$

Hence the linear velocity of any part of this subject is the vector sum of the velocity $\omega \times r_0$ due to turntable rotation and the velocity $v$ of the part with respect to the origin of the moving system

*Subcondition (1a)*

Subject is fixed in his chair

Then for all parts of his body $v = 0$ and the velocity of any part becomes $r_0 = \omega \times r_0$ Here the velocity of any point in the body depends solely on the rate of rotation of the turntable and on the distance of the point from the centre of the turntable

## 2 Basic Conditions

Subject is seated on a rotating turntable and is free to move but always faces the same direction Then he has translational motion but no rotation with respect to the inertial coordinate frame

Thus $\omega = 0$ and the velocity becomes

$$r_0 = \Omega \times R + v$$

*Subcondition (2a)*

Subject is fixed in his chair

Then $v = 0$ and $r_0 = \Omega \times R$

In this case the velocities of all points in the body depend only on the rate of rotation of the turntable and the distance from the centre of the turntable to the centre of the moving coordinate system (that is the pivot of his chair) Therefore all points in the subject move with equal velocities regardless of their distance from the centre of the turntable This means that all parts of the subject move with the same angular velocity in circles of the same radius but the axes of these circles do not necessarily coincide with one another or with the axis of the turntable

## 3 Basic Conditions

The subject is seated on a rotating turntable in a chair which rotates with respect to the rotating turntable Any point in the subject at a given instant is at a distance $r$ from the centre 0 of the *moving* axes and is rotating with angular velocity W around 0

Then the velocity of the point with respect to the moving axes system is

$$v = W \times r$$

Hence $$\mathbf{r}_0 = \mathbf{R} + \mathbf{v} + \boldsymbol{\omega} \times \mathbf{r}$$
$$= \boldsymbol{\Omega} \times \mathbf{R} + \mathbf{W} \times \mathbf{r} + \boldsymbol{\omega} \times \mathbf{r}$$
$$= \boldsymbol{\Omega} \times \mathbf{R} + (\mathbf{W} + \boldsymbol{\omega}) \times \mathbf{r}$$

**r** will be a function of both **W** and time under these conditions

For example if a point in the moving system rotates in a circle about the $Z$ axis then $\mathbf{W} = W\mathbf{k}$ since only a $Z$ component of angular velocity exists The path traced out by this point may be represented by the equation $\mathbf{r} = (r \cos Wt)\mathbf{i} + (r \sin Wt)\mathbf{j} + \ \mathbf{k}$ The velocity of the point is $\mathbf{v} = \mathbf{W} \times \mathbf{r} = -(Wr \sin Wt)\mathbf{i} + (Wr \cos Wt)\mathbf{j}$

This proves that as the point rotates around the $Z$ axis its velocity in a particular direction (such as the $X$ direction) will vary periodically from $+Wr$ to zero to $-Wr$ to zero to $+Wr$ Since Coriolis acceleration is a function of velocity the magnitude of the Coriolis acceleration at a point in a semicircular canal may change as the head rotates even when both the head and the turntable rotate at constant angular velocities

## ACCELERATION

$$\ddot{\mathbf{r}}_0 \quad \mathbf{R} + \mathbf{a} + 2\boldsymbol{\omega} \times \mathbf{v} + \boldsymbol{\alpha} \times \mathbf{r} + \boldsymbol{\omega} \times (\boldsymbol{\omega} \times \mathbf{r})$$

Here **a** is the linear acceleration of a point with respect to the moving co ordinate system and $\boldsymbol{\alpha}$ is the angular acceleration of the moving axes with respect to the inertial axes (see Appendix II page 29)

### 1 Basic Conditions

In a turntable experiment the subject is fixed in his chair (that is he is at rest with respect to the moving axes system The system however is free to move in any manner)

Then **a** and **v** are both zero and

$$\mathbf{r}_0 = \mathbf{R} + \boldsymbol{\alpha} \times \mathbf{r} + \boldsymbol{\omega} \times (\boldsymbol{\omega} \times \mathbf{r})$$

*Subcondition (1a)*

In many experiments the distance of the subject s chair from the centre of the turntable is fixed and the turntable has a constant angular velocity $\boldsymbol{\Omega}$

Then $\mathbf{R} = \boldsymbol{\Omega} \times (\boldsymbol{\Omega} \times \mathbf{R})$ that is this centrifugal acceleration acts on the subject due to rotation of the turntable

*Subcondition (1b)*

If in addition the subject faces always in the same direction so that he is translating but not rotating with respect to the inertial coordinate system then $\boldsymbol{\alpha}$ and $\boldsymbol{\omega} = 0$ and $\ddot{\mathbf{r}}_0 = \boldsymbol{\Omega} \times (\boldsymbol{\Omega} \times \mathbf{R})$ In this case the only acceleration experienced by the subject is a centrifugal one constant in magnitude for all parts of the subject but varying in direction as the turntable rotates

## 2 Basic Conditions

The subject's chair is rotating uniformly and always facing the centre of the turntable The subject is free to translate but not rotate with respect to his chair

Then $\alpha = 0$ and $\omega = \Omega$

Thus
$$\ddot{\mathbf{r}}_0 = \ddot{\mathbf{R}} + \mathbf{a} + 2\omega \times \mathbf{v} + \omega \times (\omega \times \mathbf{r})$$
$$= \Omega \times (\Omega \times \mathbf{R}) + \mathbf{a} + 2\,\Omega \times \mathbf{v} + \Omega \times (\Omega \times \mathbf{r})$$
$$= \Omega \times (\Omega \times (\mathbf{R} + \mathbf{r})) + \mathbf{a} + 2\Omega \times \mathbf{v}$$
$$= \Omega \times (\Omega \times \mathbf{r}_0) + \mathbf{a} + 2\Omega \times \mathbf{v}$$

The first term shows that the centrifugal acceleration at any point in the subject depends on the distance of the point from the centre of the turntable and on the angular velocity of the turntable In addition $\mathbf{r}_0$ will be a function of time and hence the centrifugal acceleration will also vary with time

*Subcondition* (2*a*)

The subject is not moving with respect to his chair

Then $\mathbf{a}$ and $\mathbf{v}$ are both zero and $\ddot{\mathbf{r}}_0 = \Omega \times (\Omega \times \mathbf{r}_0)$ This means that the only acceleration experienced by any point of such a subject is a centrifugal acceleration depending on the distance of the point from the centre of the turntable and on the angular velocity of the turntable Furthermore this acceleration is constant both in magnitude and in direction with respect to the subject In this case parts of the subject will experience different centrifugal forces than in the situation of Condition (1) Subcondition (*b*) where the subject's chair is translating but not rotating in space These differences will exist even if the turntable is rotating at the same angular velocity in both Condition (1) and Condition (2)

The preceding paragraphs dealt with the accelerations experienced by a uniformly rotating subject who is not moving with respect to his chair All parts of such a subject experience accelerations which will stimulate the body's linear accelerometers in particular the otoliths but will not stimulate the semicircular canals A semicircular canal and the accelerations acting on fluid molecules at different places in the canal are shown schematically in Figure 1 If the canal is rotating around 0 with angular velocity $\omega$ then the centrifugal acceleration on a fluid molecule depends on the distance from the molecule to 0 The molecules can move only around the canal hence the accelerations on molecules *C* and *D* even though they are different cannot cause fluid motion along the canal On the other hand the accelerations on *A* and *B* are in the right directions to cause fluid movement but their components along the canal are equal and try to rotate the fluid in opposite directions giving no net motion Hence centrifugal accelerations can never cause fluid motion in a semicircular canal

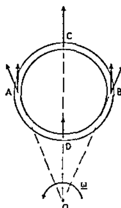



Fig 1

## 3 Basic Conditions

The subject moves either with constant acceleration **a** or with constant velocity **v** with respect to the moving coordinate system (for example, all parts of the subject move as a unit)

Then the terms **a** and $2\omega \times v$ in the acceleration equation must be considered As before, consider two cases

*Subcondition (3a)*

The subject translates uniformly around an origin but does not rotate, that is, he faces always in the same direction *in space* In this case, since the angular velocity ω of the moving axes is zero, then the term $2\omega \times v$ is always zero Hence only the term **a** the acceleration of the subject with respect to his moving coordinate system, remains in the equation

*Subcondition (3b)*

The subject always faces the centre of the rotating turntable, but is free to move otherwise

In this case, since $\omega = \Omega$ the angular velocity of the turntable (which is not zero), both **a** and $2\omega \times v$ must be considered However, since the body can translate as a unit but not rotate with respect to the moving coordinate system, then the accelerations given by the terms **a** and $2\omega \times v$ will be the same for all molecules of the endolymph and will not result in fluid movement These accelerations will affect only the otoliths the resultant effect on the otoliths will be the vector sum of terms such as gravity, Coriolis and other accelerations For uniform rotation this resultant may change in direction as seen in Condition (1) Subcondition (*b*)

Thus under any conditions in which the head moves such that the accelerations experienced by the vestibular organs are either uniform or symmetric around a semicircular canal, the endolymph will be unaffected These accelerations will affect only the otoliths

On the other hand if non symmetric accelerations around a canal occur these accelerations will cause a fluid movement and a resulting sensation of rotation The only terms in the acceleration equation which can give non symmetric effects are $\alpha \times \mathbf{r}$ and $2\omega \times \mathbf{v}$

## 4 Basic Conditions

A semicircular canal rotates about an axis with an angular acceleration $\alpha$ A schematic canal rotating about an axis through 0 is shown in Figure 2 Consider two regions $A$ and $B$ of the canal then points $A$ and $B$ in the wall of the canal will have accelerations $\alpha \times \mathbf{r}_A$ and $\alpha \times \mathbf{r}_B$ The fluid in the canal is not attached to the wall hence by its inertia the fluid will lag behind At both $A$ and $B$ this effect will tend to impart a *backwards* fluid motion relative to the wall of the canal the fluid at $A$ will have a greater backwards motion relative to the wall than the fluid at $B$ the result will be fluid rotation in the clockwise direction within the canal *opposite* to $\alpha$ This motion should be detected and interpreted by the brain as the angular acceleration $\alpha$ (The value $\mathbf{r} \times \alpha$ may then be assigned to the acceleration of a fluid molecule to give the correct relative *direction* of fluid motion as well as its theoretical magnitude ) It should be noted that the actual magnitude of cupula deflec tion will depend on the friction between the fluid and the wall on the visco sity of the fluid and on the restoring force on the cupula The situation is further complicated in that the cupula motion sensing cells require a thres hold cupula deflection before responding

For this same angular acceleration application of the equations to the other two orthogonal semicircular canals shows that no fluid motion can occur in them since the accelerations are either perpendicular to the plane of the canal or produce equal and opposing tendencies to fluid movement Hence if a canal lies precisely in a plane of rotation and is exactly at right angles to the other two canals angular acceleration in this plane is detected by one semicircular canal alone

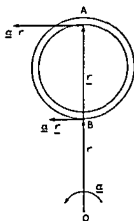



Fig 2

## 5 Basic Conditions

A semicircular canal rotates with angular velocity W about an axis. Then the linear velocity of each molecule of *both* fluid and canal wall is given by $\mathbf{v} = \mathbf{W} \times \mathbf{r}$ where $\mathbf{r}$ is the distance of the molecule from the axis. When the canal is rotating in a coordinate system which is itself rotating with angular velocity $\omega$ with respect to an inertial frame then each molecule in the canal wall will experience a Coriolis acceleration $2\omega \times \mathbf{v}$ where $\mathbf{v}$ is the linear velocity of the molecule in its own moving frame. Since the molecules in the fluid at this point are free to move around the canal they will not experience this Coriolis effect and may appear to lag behind or move opposite to the direction in which the canal wall moves provided Coriolis accelerations along the direction of the canal are not symmetric on opposite sides of the canal. In other words the endolymph will move around the canal causing a sensation of *angular acceleration* in the plane of the canal. This stimulated canal will not in general lie in either plane of rotation.

*Subcondition (5a)*

The horizontal semicircular canal $y$ rotates about an axis parallel to the axis of the turntable (Figure 3).

Then the linear velocities at $A$ and $B$ with respect to the point 0 are $\mathbf{W} \times \mathbf{r}_A$ and $\mathbf{W} \times \mathbf{r}_B$. (Even though these velocities are not equal in magnitude there is no fluid motion relative to the canal walls since the walls and the fluid are moving at the same velocity at any point.) Similarly the velocities at $C$ and $D$ will be $\mathbf{W} \times \mathbf{r}_C$ and $\mathbf{W} \times \mathbf{r}_D$ and these are equal in magnitude since $\mathbf{r}_C - \mathbf{r}_D$. In Figure 3 the angular velocity $\omega$ of the coordinate axes is parallel to the angular velocity W of the canal about the point 0. The Coriolis acceleration at $A$ is $2\omega \times \mathbf{v}_A = 2\omega \times (\mathbf{W} \times \mathbf{r}_A)$ and at $B$ is $2\omega \times \mathbf{v}_B - 2\omega \times (\mathbf{W} \times \mathbf{r}_B)$. These accelerations are unequal but will not cause fluid motion around the canal since they have no components along the canal. The Coriolis accelerations at $C$ and $D$ have components which are along the canal but these are equal and opposite hence they cannot cause fluid motion. Therefore a subject sitting on a turntable who uniformly rotates

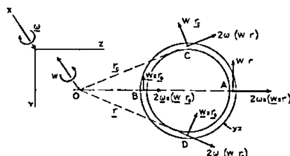



Fig 3

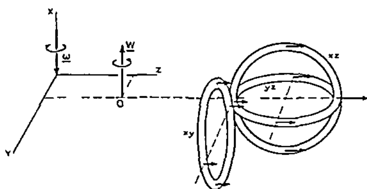



FIG 4

his head parallel to the plane of the turntable rotation will not have his horizontal canals stimulated by Coriolis accelerations

The vertical canals as well as the horizontal canal $y$ are shown schematically in Figure 4 Here again Condition (3) Subcondition ($a$) is applicable In the $xy$ vertical canal the Coriolis accelerations are everywhere at right angles to the canal hence no fluid motion can occur In the $xz$ vertical canal Figure 4 shows that the Coriolis accelerations are in the proper directions to cause fluid motion only at the top and bottom of the canal However these accelerations are equal since the top and bottom points are equidistant from 0 Again no fluid motion can occur Thus a subject sitting on a turntable who turns his head parallel to the plane of turntable rotation will not experience a sensation of angular acceleration in any plane other than the normal sensation of head motion

*Subcondition (3b)*

The subject rotates his head in a plane perpendicular to the plane of the turntable

Then the Coriolis accelerations will effectively cause some endolymph motion In Figure 5 the turntable rotates with angular velocity $\omega$ about the axis shown simultaneously the head rotates with angular velocity W about a perpendicular axis By considering each canal in turn and finding the direction of Coriolis accelerations at several points in each canal it is observed that these accelerations may cause fluid motion only in canals $xy$ and $x$ Canal $x$ is turning into the page about a vertical axis through 0 then $\mathbf{v} = \mathbf{W} \times \mathbf{r}$ of any part of the canal will point into the paper The Coriolis acceleration of the canal wall will be $2\omega \times \mathbf{v} = 2\omega \times (\mathbf{W} \times \mathbf{r})$ which will be directed vertically in the plane of the paper as shown The Coriolis acceleration will be greatest at the part of the canal wall farthest from 0 and the inertia of the fluid will then cause it to move clockwise with respect to the canal exactly as in Condition (4) The brain interprets this fluid motion as an angular acceleration in the counter clockwise direction in the plane of this canal or about an axis (parallel to the $y$ axis) at right angles to both W

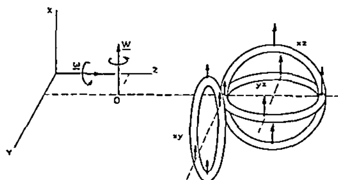



Fig 5

and ω If the angular velocity ω represents a counter clockwise rotation of the turntable, then the angular velocity W represents the motion involved in nodding the head forward while facing the centre of the turntable the resultant stimulation of the $xz$ canal is interpreted as a tumbling or angular acceleration of the head to the right The Coriolis accelerations along the canal $xy$ may be shown by the same analysis to be equal on opposite sides of the canal This means that no fluid motion occurs in this canal at the position shown in Figure 5

The value of the Coriolis acceleration $2\omega \times v$ is numerically equal to $2\omega v \sin\theta$ where $\theta$ is the smaller angle between the vectors ω and v In Figure 5 the velocity v of each molecule in the $xz$ canal is at right angles to ω hence $\theta = 90°$, $\sin\theta = 1$ and the Coriolis acceleration is a maximum When the canals have turned through 90° as in Figure 6, there will be no endolymph acceleration in canal $xz$ since then $\sin\theta = 0$ for all parts of this canal The other vertical canal $xy$ will experience no Coriolis acceleration at the instant that the acceleration in canal $xz$ is a maximum, but will experience an increasing stimulation as the stimulation of $xz$ dies away In general for rotation in the plane of one canal the canals in the other two planes will both be stimulated in a sinusoidal manner with one canal receiving its maximum

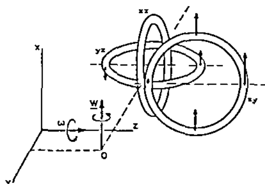



Fig 6

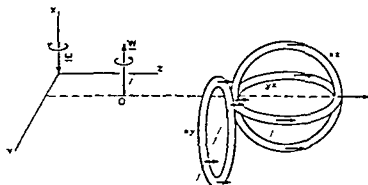



Fig 4

his head parallel to the plane of the turntable rotation will not have his horizontal canals stimulated by *Coriolis accelerations*

The vertical canals as well as the horizontal canal $yz$ are shown schematically in Figure 4 Here again Condition (5) Subcondition ($a$) is applicable In the $xy$ vertical canal the Coriolis accelerations are everywhere at right angles to the canal hence no fluid motion can occur In the $xz$ vertical canal Figure 4 shows that the Coriolis accelerations are in the proper directions to cause fluid motion only at the top and bottom of the canal However *these accelerations are equal since the top and bottom points are equidistant* from 0 Again no fluid motion can occur Thus a subject sitting on a turntable who turns his head parallel to the plane of turntable rotation will not experience a sensation of angular acceleration in any plane other than the normal sensation of head motion

*Subcondition* (5$b$)

The subject rotates his head in a plane perpendicular to the plane of the turntable

*Then the Coriolis accelerations will effectively cause some endolymph* motion In Figure 5 the turntable rotates with angular velocity $\omega$ about the axis shown simultaneously the head rotates with angular velocity W about a perpendicular axis By considering each canal in turn and finding the direction of Coriolis accelerations at several points in each canal it is observed that these accelerations may cause fluid motion only in canals $xy$ and $xz$ Canal $xz$ is turning into the page about a vertical axis through 0 then $\mathbf{v} = \mathbf{W} \times \mathbf{r}$ of any part of the canal will point into the paper The Coriolis acceleration of the canal wall will be $2\omega \times \mathbf{v} = 2\omega \times (\mathbf{W} \times \mathbf{r})$ which will be directed vertically in the plane of the paper, as shown The Coriolis acceleration will be greatest at the part of the canal wall farthest from 0 and the inertia of the fluid will then cause it to move clockwise with respect to the canal exactly as in Condition (4) The brain interprets this fluid motion as an angular acceleration in the counter-clockwise direction in the plane of this canal about an axis (parallel to the $y$ axis) at right angles to both W

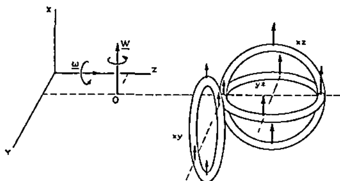



Fig 5

and ω If the angular velocity ω represents a counter clockwise rotation of the turntable, then the angular velocity W represents the motion involved in nodding the head forward while facing the centre of the turntable, the resultant stimulation of the $xz$ canal is interpreted as a tumbling or angular acceleration of the head to the right The Coriolis accelerations along the canal $xy$ may be shown by the same analysis to be equal on opposite sides of the canal This means that no fluid motion occurs in this canal at the position shown in Figure 5

The value of the Coriolis acceleration $2\omega \times v$ is numerically equal to $2\omega v \sin\theta$, where $\theta$ is the smaller angle between the vectors ω and v In Figure 5 the velocity v of each molecule in the $xz$ canal is at right angles to ω, hence $\theta = 90°$, $\sin\theta = 1$ and the Coriolis acceleration is a maximum When the canals have turned through 90° as in Figure 6, there will be no endolymph acceleration in canal $xz$, since then $\sin\theta = 0$ for all parts of this canal The other vertical canal $xy$ will experience no Coriolis acceleration at the instant that the acceleration in canal $xz$ is a maximum but will experience an increasing stimulation as the stimulation of $xz$ dies away In general for rotation in the plane of one canal, the canals in the other two planes will both be stimulated in a sinusoidal manner, with one canal receiving its maximum

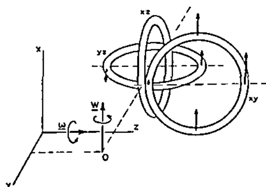



Fig 6

stimulation when the other receives its minimum However, the vector sum of their stimuli will be constant in magnitude

It has been assumed in the preceding analysis that the semicircular canals were symmetric about the axis of rotation In fact this may not be true the *axis of rotation may not coincide with an axis of symmetry of any canal* It may be shown however that this will affect only the *magnitude* of the Coriolis accelerations in any canal at a given time the sinusoidal character of the stimulation described in the previous paragraph will still hold and the total stimulus will be the same For this reason the semicircular canals may be represented in any situation by three orthogonal canals with one in the plane of rotation of the head

The Coriolis accelerations affect not only the endolymph causing a sensation of rotational acceleration but also stimulate the otoliths The subjective sensation will be a combination of rotational acceleration and translation The direction and magnitude of the otolith stimulation is easily calculated from the general equations of motion derived in Appendix II page 29

## FORCES ON THE VESTIBULAR APPARATUS IN A HUMAN CENTRIFUGE EXPERIMENT

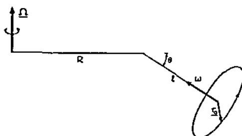
The calculation of the forces acting on the vestibular apparatus during any rotational experiment may be complex but can be illustrated by a typical example

A diagrammatic representation of a human centrifuge experiment is shown in Figure 7 The centrifuge arm rotates in the horizontal plane with constant angular velocity $\Omega$ a gondola containing a subject is pivoted about the arm at a distance $R$ from the centrifuge axis the vestibular apparatus of the subject is at a distance $l$ from the pivot of the gondola During rotation of the centrifuge the gondola assumes a constant angle $\theta$ with respect to the horizontal The subject rotates his head with angular velocity $\omega$ about an axis coincident with the direction of $l$

The centrifugal acceleration affecting only the otoliths is easily calculable It will consist of two vector components one due to rotation of the arm directed away from the centre in the plane of the rotation and the other due to head rotation by the subject The latter component will normally be negligible in comparison with the former

Hence the magnitude of the centrifugal acceleration will be approximately equal to $(R + l\cos\theta)\Omega^2$

The Coriolis acceleration caused by the subject s head rotation will now be calculated The angular velocity of the head rotation $\omega$ can be resolved into two vectors—one parallel to the plane of the centrifuge and one perpendicular to it The former will induce no Coriolis acceleration effects in




the semicircular canals hence only the latter need be considered Consider Figure 8 where $r_0$ is the radius of rotation of the canals about the central axis through the head $\omega$ is the angular velocity of this rotation and $\Omega$ is the angular velocity of centrifuge rotation (The plane of rotation of the head is *actually perpendicular to the plane of the paper ) Three semicircular canals* are shown schematically these will move out of the plane of the paper at the lower left and into the paper at the upper right A parallel beam of light shining on this figure from the left will project a shadow of the canals on a plane to the right of the figure If the plane is parallel to the direction of $\Omega$ and perpendicular to the plane of the paper then this shadow will be a projection of the motion of the canals in the plane to be considered since motions in this plane will be perpendicular to the plane of the centrifuge and will result in Coriolis effects

The canals shown in the diagram may be chosen in orientation such that one canal (canal 3) will give a constant elliptical projection on the projection plane The three canals may be assumed to be arbitrarily oriented in space since the brain appears to give the proper interpretation of acceleration in

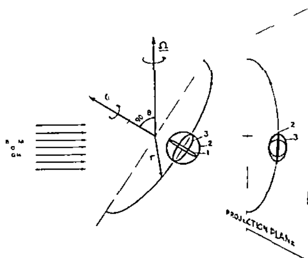



Fig 8

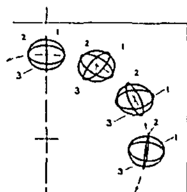


FIG. 9

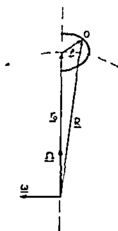


FIG. 10

space regardless of the initial head orientation. The other two canals (1 and 2) will be orthogonal to the projection plane such that the projection of canal 1 on this plane will be an ellipse when that of canal 2 is a straight line; these projections will each change continuously into a straight line from an ellipse and into an ellipse from a straight line during every rotation of the head through 90° (Figure 9).

Assuming that the brain integrates the responses from the semicircular canals to give the proper magnitude and direction of the total response, then it must add vectorially the output from these two canals. That is, accelerations from the two canals are added to give the proper magnitude and direction with respect to the head of the total angular acceleration of the vestibular apparatus.

Now since canals 1 and 2 are orthogonal, the stimulation in canal 1 will be a maximum when that in canal 2 is zero. At this instant, the magnitude and direction of the resultant will be identically the magnitude and direction of the stimulus in canal 1, and the calculation of this stimulus therefore provides the required information.

Consider a canal projection as shown in Figure 10. Here the canal at this instant is rotating into the plane of the paper, that is, its velocity is at right angles to $\mathbf{\Omega}$. Then $\mathbf{v}$, the velocity of the point 0, is

$$\begin{aligned}\mathbf{v} &= \boldsymbol{\omega} \times \mathbf{R} \\ &= \boldsymbol{\omega} \times (\mathbf{r}_0 + \boldsymbol{\rho}) \\ &= (\boldsymbol{\omega} \times \mathbf{r}_0) + (\boldsymbol{\omega} \times \boldsymbol{\rho})\end{aligned}$$

Now $\boldsymbol{\omega} \times \mathbf{r}_0$ is constant over the entire canal; therefore this term produces no movement of the endolymph. This is equivalent to putting the radius $\mathbf{r}_0$ equal to zero. Hence the diagram can be simplified as shown in Figure 11. The velocity of 0 into the paper has the magnitude $\omega y$ where $y$ is the component of $\boldsymbol{\rho}$ in the Y direction. The acceleration at 0 is $C_A = 2\Omega\omega y$ and will be

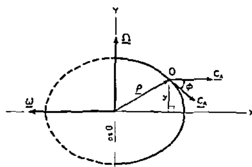



Fig 11

parallel to the X axis. However, the part of $C_A$ effective in causing fluid movement is the component along the canal

$$C_A' = C_A \cos\phi$$

Now $\quad \tan\phi = \frac{dy}{dx} \qquad \cos\phi = \frac{1}{\sqrt{1 + \tan^2\phi}}$

$$\cos\phi = \frac{1}{\sqrt{1 + \left(\frac{dy}{dx}\right)^2}}$$

Now the force $dF$ on a fluid mass $dm$ in the canal is

$$dF = C_A' \, dm$$

also $\quad dm = A d_0 \, ds$

where $ds$ = incremental length along the canal circumference, $A$ = cross sectional area of canal, and $d_0$ = density of endolymph.

Also $\quad ds = \sqrt{1 + \left(\frac{dy}{dx}\right)^2}\, dx$

Hence $\quad dF = C_A' \, dm = C_A \cos\phi \, A d_0 \, ds$

$$= 2\Omega\omega y \, A d_0 \, dx$$

Then by integration the force on a diaphragm in the canal is

$$F = \int_0 2\Omega\omega \, A d_0 \, y \, dx$$

$$= 2 A d_0 \Omega\omega \int_0^r y \, dx$$

The projected canal was an ellipse with semi-major axis $= r$, semi-minor axis $= r\cos\theta$, and is represented by the equation

$$\frac{x^2}{r^2} + \frac{y^2}{r^2\cos^2\theta} = 1$$

or

$$y = (\cos\theta)\sqrt{r^2 - x^2}$$

Therefore

$$F = 8 d_0 A\Omega\omega \int_0^r (\cos\theta)\sqrt{r^2 - x^2}\, dx$$

$$= 8 d_0 A\Omega\omega\cos\theta\left(\frac{1}{2}\left[x\sqrt{r^2 - x^2} + r^2 \arcsin\frac{x}{r}\right]_0^r\right)$$

$$= 4 d_0 A\Omega\omega\cos\theta\,\frac{\pi r^2}{2}$$

$$= 2\pi d_0 A\Omega\omega r^2\cos\theta$$

If $\omega$ is known as a function of time, then this force $F$ can be included in a differential equation for cupula deflection as a function of time and the equation solved.

## FURTHER PROBLEMS IN ROTATIONAL MOTION

The application of the laws of mechanics discussed in this report to the inner ear under different conditions of motion leads to several predictions. For example, it is well known that in rotational motion the phenomenon of nystagmus occurs about the same axis as that about which the body is or appears to be accelerated. From the preceding analysis the direction and magnitude of the nystagmus may be predicted from the knowledge of the movements involved. If, for example, a subject is seated on a chair always facing the centre of a rotating centrifuge and simultaneously is rotated bodily at a uniform rate about an axis from front to back (for example, his head may be rotated down and to his right) then the following phenomenon should occur (assuming that nystagmus is not persistent). If the subject at the instant considered is in the upright position, semicircular canals are then being stimulated to give the subject a sensation of angular acceleration either forward on his face or backwards, depending on the direction of turntable rotation. At this instant his eyes will therefore oscillate about this axis, moving up and down in a vertical plane in their sockets. When the subject has been rotated through 90° (and is lying on his right side) *other* semicircular canals will be stimulated—those now in *another* vertical plane in fixed space. His eyes will then oscillate in this vertical fixed space plane, or eye movement is *from side to side*. Thus the direction of the nystagmus should rotate smoothly through 90° *with respect to the eye sockets* as the man is rotated. It should be possible to obtain experimental

verification of this point provided conditions are chosen such that the persistence of nystagmus is unimportant or eliminated

Perhaps the major problem in vestibular physiology at the moment is the prediction and explanation of the effects on the human body of various types of motion In rotational motion the deflection of cupulae in the semicircular canals is a function of the stimuli applied to the canals A current mathematical model of the inner ear leads to a second order linear differential equation for this deflection as a function of time The predictions of this model agree qualitatively with observations made under various experimental conditions in addition cupula deflection can be predicted quantitatively from this model as a function of stimulus Until fairly recently no measure of this deflection was known except indirectly from the associated nystagmus This was unreliable however in some circumstances Present work in this laboratory has indicated other potential methods of accurately assessing body stimulation following acceleration and rotation on a turntable Since the magnitude of cupula deflection appears to be directly calculable from a knowledge of the rotational stimuli a reliable experimental determination of this deflection or of a physiological effect proportional to it is essential in predicting the stimulation from any rotational motion imposed on the subject In turn this would greatly assist the formulation of a theory of motion sickness and disorientation

## SUMMARY

In this report equations of motion are applied to the human vestibular apparatus when undergoing movement Typical turntable experiments are considered and the physical effects of the associated motions are analyzed with special emphasis on the forces experienced by the semicircular canals In appendices to the report vector algebra is developed and equations of motion are derived in vector form Graphs and a nomograph for evaluation of the results of centrifugal acceleration are presented in another appendix

Vestibular physiology is a field of vital importance in modern scientific endeavour if man is expected to operate high speed aircraft and space vehicles efficiently The description and analysis of the forces and motions encountered is the task of the physicist but the experimental work on the human subject in the laboratory and the reactions observed in the field must be evaluated by the physiologist The most rapid and fruitful advances in this area will undoubtedly result from the collaborative efforts of physicists and physiologists

## ACKNOWLEDGEMENTS

The advice and assistance of Mr J F Kennedy and Dr I H Turl of this Laboratory has been invaluable in the preparation of this report Helpful advice and encouragement have also been received from Dr K F Money Dr I E MacHattie of this Laboratory and W/C R A Stubbs of IAM/OMF

# VECTOR ALGEBRA

Physical quantities are normally classified as either scalars or vectors. A quantity which can be fully specified in terms of a given number of units is a *scalar*, written as $R$; a *vector* is specified fully by a number of units with an associated direction, and written as **R**. Speed is a scalar; if a car is travelling at 50 m.p.h., its *speed* is 50 m.p.h. On the other hand, velocity is a vector; if the car is travelling north at this rate, its *velocity* is 50 m.p.h. northward. Similarly, *distance* is a scalar, while *displacement* is the corresponding vector. For example, if a man leaves a place and walks 5 miles, he has travelled *a distance of 5 miles*; *if he walks to the north*, his *displacement* is 5 miles northward.

A vector may be depicted by an arrow drawn from an origin with its length representing the magnitude of the vector and its direction representing the direction of the vector.[1] Figure 12, page 23, illustrates a vector **R** of length 3 units directed to the north-east; this might represent the velocity of a car travelling north-east at 30 m.p.h. This diagram shows that the car is moving *north* and *east* at the same time. Generally, any vector can be resolved into components which are at an angle to each other. To simplify notation, the *magnitude* of a vector such as **R** will be written as $R$, i.e. italics will be used. From trigonometry, the magnitude of the velocity component to the north is

$$R_N = R \sin\theta$$

and the velocity component east is

$$R_E = R\cos\theta$$

where $R$ is the magnitude or number of units in the vector **R**. Since these components are at right angles, then by Pythagoras' theorem

$$R_N^2 + R_E^2 = R^2$$

and by substitution this can be verified as follows:

$$\begin{aligned} R_N^2 + R_E^2 &= (R\sin\theta)^2 + (R\cos\theta)^2 \\ &= R^2(\sin^2\theta + \cos^2\theta) \\ &= R^2 \end{aligned}$$

[1] In this work illustrations will be represented in a Cartesian coordinate system, i.e. all the axes are at right angles to one another.

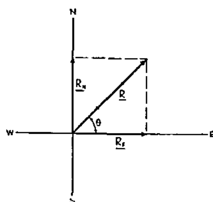



Fig 12

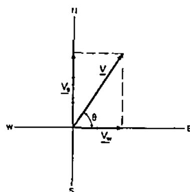



Fig 13

Conversely, if the components $R_N$ and $R_E$ of the vector **R** are known or can be calculated, the direction of **R** can be determined from these components. From the first two equations above

$$\frac{R_N}{R_E} = \frac{R\sin\theta}{R\cos\theta}$$

$$= \frac{\sin\theta}{\cos\theta}$$

$$= \tan\theta$$

then the direction of the vector **R** is given by $\theta = \tan^{-1} R_N/R_E$ (that is, $\theta$ is the angle whose tangent is $R_N/R_E$).

It is quite common to have more than one vector referred to the same origin. Consider a bird flying with a velocity of 30 m.p.h. *north*; at the same time a wind of 20 m.p.h. is blowing towards the *east*. Hence the bird is blown east with a velocity of 20 m.p.h. and is also travelling north at 30 m.p.h. This is shown in Figure 13 by $V_B$ representing the bird's flying velocity and $V_W$ his wind drift velocity. Since these two *component* velocities are at right angles to each other, the magnitude of his *total* velocity $V$, as seen by an observer on the ground, will be

$$V^2 = V_B^2 + V_W^2$$

$$= 30^2 + 20^2$$

$$= 1300$$

or

$$V = \sqrt{1300} = 36.1 \text{ m.p.h.}$$

Now the direction of V (that is, the direction in which the bird travels with respect to the ground) is given by

$$\theta = \tan^{-1}\frac{V_B}{V_W}$$

$$= \tan^{-1}\frac{30}{20} = \tan^{-1} 1.5$$

$$= 56^\circ 19'$$

The bird's flight is fully specified by stating that he has a velocity of 36.1 m.p.h. 56°19' north of east.

In the preceding example the *components* of velocity to be added (the bird's velocity north and the wind velocity east) were at right angles and easily added. Vectors, whether at right angles or not, can be added in two ways: (1) exactly by finding components, and (2) approximately by graphical means.

To add vectors by method (1) consider Figure 14. Here $V_B$, the velocity of the bird, is 30 m.p.h. in a direction 60° north of east, and $V_W$, the velocity of the wind, is 20 m.p.h. in a direction 30° north of east. Each velocity can be resolved into two components at right angles, one component towards the north and one towards the east. Consider $V_B$; then the north component is given by $V_B \sin 60^\circ = 26$ m.p.h. north. The east component is $V_B \cos 60^\circ = 15$ m.p.h. east. Similarly, the north component of $V_W$ is $V_W \sin 30^\circ = 10$ m.p.h. north and the east component is $V_W \cos 30^\circ = 17.3$ m.p.h. east. Since the north components of $V_B$ and $V_W$ are in the same direction, then the total north component is just their algebraic sum: $V_N = 26 + 10 = 36$ m.p.h. north. Similarly, the total east component is $V_E = 15 + 17.3 = 32.3$ m.p.h. east. Hence the magnitude of the resultant of these two velocities of 36 m.p.h. north and 32.3 m.p.h. east is given by

$$V^2 = V_N^2 + V_E^2$$

$$= 36^2 + 32.3^2$$

$$= 1296 + 1043$$

$$= 2339$$

or

$$V = \sqrt{2339}$$

$$= 48.4 \text{ m.p.h.}$$

The direction of this resultant is $\theta = \tan^{-1} 36/32.3 = \tan^{-1} 1.113 = 48^\circ 4'$ north of east.

Any number of vectors acting at a point can be added by extending this procedure of method (1).

Method (2) is a faster but less accurate graphical means of adding vectors. Consider again Figure 14; here the following procedure may be used to obtain the resultant $V$. A new $V_B$ is drawn in the same direction and of the same length as the previous $V_B$, but the tail of its arrow is placed at the head of the other vector $V_W$. Then a line drawn from the tail of $V_W$ to the head of the new $V_B$ represents the magnitude and direction of the resultant $V$. It is ap-

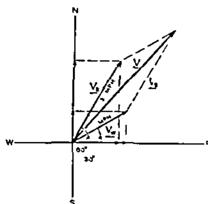



FIG 14

parent that this leads to the same resultant as method (1) The magnitude and direction of V can be found with a ruler and protractor

This process can be extended to include any number of vectors for if two vectors are added graphically to give a resultant $V_1$ then a third vector can be added to $V_1$ to give another resultant $V_2$ and so on In fact the intermediate resultant may be omitted The general procedure then is to draw all the vectors tail to head giving each vector its appropriate magnitude and direction and joint the tail of the first to the head of the last to obtain the final resultant

As well as vectorial addition three forms of multiplication are defined for vectors Vectors may be multiplied by a scalar (that is a number) quite simply $3R = 3$ times R Scalar multiplication changes only the magnitude of R (and its components) but not its direction This multiplication is distributive that is $3(R_1 + R_2) = 3R_1 + 3R_2$ This leads to the useful concept of unit vectors these are vectors of length one unit and direction along an axis of the coordinate system For example in Figure 12 page 23 unit vectors might be chosen as **i** and **j** where **i** is a vector of length one unit (such as one m p h ) in the east direction and **j** is one unit length in the north direction Then any vector or vector component in these directions may be written as a scalar times the unit vector thus $R_E = R_E i$ and $R_N = R_N j$

The other types of vector multiplication involve the product of two or more vectors One type leads to a scalar resultant the other leads to a new vector A most useful and common physical concept is that of work if a force is applied to a body and causes a displacement then the work done on the body is the product of the displacement and the force in the direction of motion Work is a scalar but force and displacement are both vectors Hence in Figure 15 if a toboggan is pulled along the ground by a force **F** applied for a distance **r** then the work done is $r$ times $(F \cos \theta)$ since $(F \cos \theta)$ is the component of the force **F** in the direction of motion **r** The work is given by

$$W = rF \cos \theta$$

$$\theta = \tan^{-1}\frac{V_B}{V_W}$$

$$= \tan^{-1}\frac{30}{20} = \tan^{-1} 1.5$$

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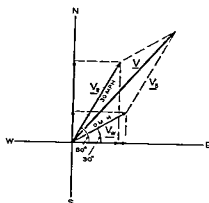



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$$W \quad rF \cos \theta$$




FIG 15

and is conventionally written as

$$W = \mathbf{r}\ \mathbf{F}$$

Here **r** **F** is called the *scalar* or *dot product* of the vectors **r** and **F** and **r** **F** is equivalent to the scalar of magnitude $rF\cos\theta$ The dot product is distributive i e

$$\mathbf{A}\ (\mathbf{B}+\mathbf{C}) = \mathbf{A}\ \mathbf{B} + \mathbf{A}\ \mathbf{C}$$

and commutative

$$\mathbf{A}\ \mathbf{B} = \mathbf{B}\ \mathbf{A}$$
$$= AB\cos\theta$$

If **A** and **B** are vectors in a two dimensional Cartesian coordinate system then their dot product may be written in terms of unit vectors **i** along the X axis and **j** along the Y axis as

$$\mathbf{A}\ \mathbf{B} = (A_x\mathbf{i} + A_y\mathbf{j})\ (B_x\mathbf{i} + B_y\mathbf{j})$$
$$A_xB_x\mathbf{i}\ \mathbf{i} + A_xB_y\mathbf{i}\ \mathbf{j} + A_yB_x\mathbf{j}\ \mathbf{i} + A_yB_y\mathbf{j}\ \mathbf{j}$$

where $A_x$ $A_y$ $B_x$ and $B_y$ are magnitudes of the components of **A** and **B** along the X and Y axes

Now $\mathbf{i}\ \mathbf{i} - i^2(\cos 0°) = 1$ and similarly $\mathbf{j}\ \mathbf{j}\quad 1$

and $\mathbf{i}\ \mathbf{j}\quad \mathbf{j}\ \mathbf{i} = ij(\cos 90°) = 0$

then $\mathbf{A}\ \mathbf{B} = A_xB_x + A_yB_y$ which is a scalar

The third type of vector multiplication is the vector or cross product This again arises naturally from physical ideas If a charged particle such as an electron is injected with velocity **V** into a magnetic field which makes some angle with the path of the electron then the electron will start to deviate from its original straight line path This deviation will be at right angles to *both the magnetic field and the direction of velocity of the electron* Since the velocity is being changed then a force is being applied to the electron This force may be represented by a vector **F** the velocity of the electron by the vector **V** and the magnetic field since it has both magnitude and direction by a vector **B** Experiments show that the force on the electron is equal to the charge on the electron *e* times the product of the electron velocity V the magnetic field *B* and the sine of the angle $\theta$ between **V** and **B** that is equal to $B\sin\theta$) The direction of the force is the same as the direction of advan he point of a right hand screw when turned through the smaller

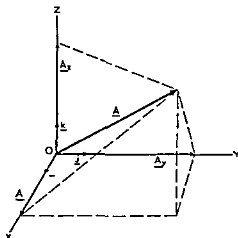



Fig 16

angle from **V** to **B** Hence both magnitude and direction of **F** are given by $\mathbf{F} = e(\mathbf{V} \times \mathbf{B})$ where $\mathbf{V} \times \mathbf{B}$ is called the vector product of **V** and **B** The direction of **F** is given by the right hand screw rule the magnitude of **F** by the product of $e$ $V$ $B$ and $\sin\theta$ where $\theta$ is the smaller angle between **V** and **B** This cross product is distributive that is $\mathbf{A} \times (\mathbf{B} - \mathbf{C})$ $\mathbf{A} \times \mathbf{B} + \mathbf{A} \times \mathbf{C}$ but not commutative $\mathbf{A} \times (\mathbf{B} \times \mathbf{C})$ is not equal to $\mathbf{B} \times (\mathbf{A} \times \mathbf{C})$

In Figure 16 a three dimensional Cartesian coordinate system is shown Here the $X$ $Y$ and $Z$ axes are orthogonal or at right angles to one another **i** **j** and **k** are unit vectors in the $X$ $Y$ and $Z$ directions respectively If **A** is a vector in space the orthogonal components $\mathbf{A}_x$ $\mathbf{A}_y$ and $\mathbf{A}_z$ of **A** can be found by dropping perpendiculars from **A** to the $X$ $Y$ and $Z$ axes Then $\mathbf{A}_x$ $A_x\mathbf{i}$ $\mathbf{A}_y = A_y\mathbf{j}$ and $\mathbf{A}_z$ $A_z\mathbf{k}$

Then the cross product of **A** and **B** may be expanded in terms of vector components as

$$\begin{aligned}\mathbf{A} \times \mathbf{B} &= (A_x\mathbf{i} + A_y\mathbf{j} + A_z\mathbf{k}) \times (B_x\mathbf{i} + B_y\mathbf{j} + B_z\mathbf{k}) \\ &= A_xB_x(\mathbf{i} \times \mathbf{i}) + A_xB_y(\mathbf{i} \times \mathbf{j}) + A_xB_z(\mathbf{i} \times \mathbf{k}) \\ &\quad + A_yB_x(\mathbf{j} \times \mathbf{i}) + A_yB_y(\mathbf{j} \times \mathbf{j}) + A_yB_z(\mathbf{j} \times \mathbf{k}) \\ &\quad + A_zB_x(\mathbf{k} \times \mathbf{i}) + A_zB_y(\mathbf{k} \times \mathbf{j}) + A_zB_z(\mathbf{k} \times \mathbf{k})\end{aligned}$$

By reference to Figure 16 $\mathbf{i} \times \mathbf{i} = \mathbf{j} \times \mathbf{j}$ $\mathbf{k} \times \mathbf{k} = 0$ (since $\sin\theta = \sin 0° = 0$) and $\mathbf{i} \times \mathbf{j}$ $\mathbf{k} = -\mathbf{j} \times \mathbf{i}$ etc

Hence $\mathbf{A} \times \mathbf{B} = (A_yB_z - A_zB_y)\mathbf{i} + (A_zB_x - A_xB_z)\mathbf{j} + (A_xB_y - A_yB_x)\mathbf{k}$ which is a vector

The rate of change with time of a vector is required that is

$$\begin{aligned}\dot{\mathbf{A}} &= \frac{d}{dt}(\mathbf{A}) \\ &= \frac{d}{dt}(A_x\mathbf{i} + A_y\mathbf{j} + A_z\mathbf{k})\end{aligned}$$

which by ordinary rules of calculus for derivatives of products is

$$\dot{\mathbf{A}} = \frac{dA_x}{dt}\mathbf{i} + A_x\frac{d\mathbf{i}}{dt} + \frac{dA_y}{dt}\mathbf{j} + A_y\frac{d\mathbf{j}}{dt} + \frac{dA_z}{dt}\mathbf{k} + A_z\frac{d\mathbf{k}}{dt}$$

If the unit vectors do not change with time, as would be the case if the coordinate system was fixed to the earth, then

$$\frac{d\mathbf{i}}{dt} = 0 \quad \text{etc.} \quad \text{and}$$

$$\dot{\mathbf{A}} = \dot{A}_x\mathbf{i} + \dot{A}_y\mathbf{j} + \dot{A}_z\mathbf{k}$$

If **A** is the displacement of a point, then $\dot{\mathbf{A}}$ is the time rate of change of this displacement or the velocity of the point. Similarly the acceleration is the time rate of change of this velocity, or

$$\ddot{\mathbf{A}} = \ddot{A}_x\mathbf{i} + \ddot{A}_y\mathbf{j} + \ddot{A}_z\mathbf{k}$$

All vectors considered so far have been *linear* vectors; an *axial* vector is associated with rotation. *If a vector makes an angle θ with a reference* axis, then the time rate of change of this angle is $d\theta/dt = \dot{\theta} = \omega$; this is called the *angular speed* of the vector. Then the *angular acceleration* is $d\omega/dt = \dot{\omega} = \ddot{\theta} = \alpha$. It can be shown that the linear speed of a point on the vector is $V = r\omega$, where $r$ is the distance of the point from the axis of rotation. Similarly the magnitude of the linear acceleration of the point is $a = r\alpha$. Consider a circle turning with angular velocity $\omega$ about its centre. If the radius of the circle is $r$, the magnitude of the velocity $V$ of a point on the circumference of the circle is $V = r\omega$. Angular velocity can be represented by a vector $\boldsymbol{\omega}$ drawn along the axis of rotation. The length of $\boldsymbol{\omega}$ is determined by the magnitude of the angular velocity; the *direction* of $\boldsymbol{\omega}$ is determined by the direction of advance of a right hand thread screw point when the screw is turned in the direction of rotation. Now **V** is a tangential velocity—it is at right angles to **r**. It is also at right angles to the axis of rotation. Hence **V** can be written as a cross product of vectors $\boldsymbol{\omega}$ and **r**; in fact the correct formula is $\mathbf{V} = \boldsymbol{\omega} \times \mathbf{r}$. Similarly the tangential acceleration **a** is the vector product of $\boldsymbol{\alpha}$ and **r**, that is $\mathbf{a} = \boldsymbol{\alpha} \times \mathbf{r}$.

# ROTATIONAL MOTION

Consider the coordinate systems shown in Figure 17 where the $0_0$ co ordinate frame is inertial (space fixed) and the 0 coordinate system moves with respect to it Then the position of a point $P$ can be specified with respect to the inertial frame by the vector $\mathbf{r}_0$ Here $\mathbf{r}_0$ is the vector sum of two com ponents the vector $\mathbf{R}$ which is the position of the origin 0 with respect to the inertial frame and the vector $\mathbf{r}$ which is the position of $P$ with respect to the moving coordinate frame

That is $\qquad \mathbf{r}_0 \quad \mathbf{R} + \mathbf{r}$

The velocity of $P$ is then $\qquad \mathbf{r}_0 = \mathbf{R} + \mathbf{r}$

The vector $\mathbf{R}$ is the velocity of the origin 0 with respect to the inertial frame To evaluate the term $\mathbf{r}$ it may be written as

$$\mathbf{r} = \frac{d}{dt}(x\mathbf{i} + y\mathbf{j} + \ \mathbf{k})$$

where $x$ $y$ and are components of $\mathbf{r}$ along the X Y and $Z$ axes and $\mathbf{i}$ $\mathbf{j}$ and $\mathbf{k}$ are unit vectors in these directions

Then $$\mathbf{r} - x\mathbf{i} + y\mathbf{j} + \ \mathbf{k} + x\frac{d\mathbf{i}}{dt} + y\frac{d\mathbf{j}}{dt} + \ \frac{d\mathbf{k}}{dt}$$

Now $$x\mathbf{i} + y\mathbf{j} + \ \mathbf{k} = \mathbf{v}$$

the linear velocity of $P$ in the moving frame

To evaluate terms of the type $x(d\mathbf{i}/dt)$ consider Figure 18

Suppose the moving coordinate system is rotating with angular speed $\omega$ then $\omega$ will be made up of three components $\omega_x$ $\omega_y$ and $\omega_z$ the rates of rotation about the X Y and $Z$ axes respectively

As the coordinate system rotates in a time $\Delta t$ the unit vector $\mathbf{i}$ changes in direction to a new unit vector $\mathbf{i} + \Delta\mathbf{i}$ Consider rotation of the vector $\mathbf{i}$ about the Y axis Then in a time $\Delta t$ the vector will turn through an angle $\theta \quad \omega_y \Delta t$ (The minus sign appears because counter clockwise rotations are conventionally taken as positive ) Now the linear distance travelled by the head of the vector $\mathbf{i}$ will be $i\theta - \omega_y \Delta t i$ since the length of the arc swept out by a radius of a circle is equal to the angle turned through (expressed in radians) times the radius If $\Delta t$ is very short and thus $\theta$ is a very small angle then the arc swept out will be practically a straight line parallel to the Z axis Hence the change in the vector $\mathbf{i}$ caused by a rotation about the Y axis

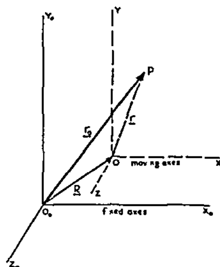



Fig 17

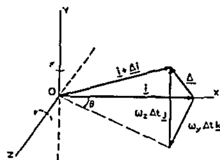



Fig 18

will be obtained by adding to $\mathbf{i}$ a vector of magnitude $-\omega_y \Delta t 1 = -\omega_y \Delta t$ in the $Z$ direction that is a vector $(-\omega_y \Delta t)\mathbf{k}$

Similarly, if $\mathbf{i}$ rotates about the $Z$ axis the vector to be added to $\mathbf{i}$ becomes $(+\omega_z \Delta t)\mathbf{j}$ Rotation about the $X$ axis produces no change in $\mathbf{i}$ Hence the total change in $\mathbf{i}$ is given by the vector sum $\Delta \mathbf{i} = \omega_z \Delta t \mathbf{j} - \omega_y \Delta t \mathbf{k}$ as shown in Figure 18 page 30

Then

$$\frac{\Delta \mathbf{i}}{\Delta t} = \omega_z \mathbf{j} - \omega_y \mathbf{k}$$

But $\Delta \mathbf{i}/\Delta t$ = time rate of change of $\mathbf{i} = d\mathbf{i}/dt$ Similarly, $d\mathbf{j}/dt$ and $d\mathbf{k}/dt$ can be found Now the last terms of the expression for $\mathbf{r}$ were

$$x\frac{d\mathbf{i}}{dt} + y\frac{d\mathbf{j}}{dt} + z\frac{d\mathbf{k}}{dt}$$

To evaluate these consider the cross product $\boldsymbol{\omega} \times \mathbf{r}$

$$\boldsymbol{\omega} \times \mathbf{r} = (\omega_x \mathbf{i} + \omega_y \mathbf{j} + \omega_z \mathbf{k}) \times (x\mathbf{i} + y\mathbf{j} + z\mathbf{k})$$

Multiplying and collecting terms,

$$\boldsymbol{\omega} \times \mathbf{r} = x(\omega_z \mathbf{j} - \omega_y \mathbf{k}) + y(\omega_x \mathbf{k} - \omega_z \mathbf{i}) + z(\omega_y \mathbf{i} - \omega_x \mathbf{j})$$
$$= x\frac{d\mathbf{i}}{dt} + y\frac{d\mathbf{j}}{dt} + z\frac{d\mathbf{k}}{dt}$$

Here $\boldsymbol{\omega}$ is the rate of rotation of the moving axes with respect to the inertial $(O_0)$ axes

Then finally $\mathbf{r} = \mathbf{v} + \boldsymbol{\omega} \times \mathbf{r}$ and $\mathbf{r}_0 = \mathbf{R} + \boldsymbol{\omega} \times \mathbf{r} + \mathbf{v}$

Here $\mathbf{r}_0$ is the velocity of a point relative to a space fixed or inertial frame This is composed of $\mathbf{R}$ the velocity of the origin of the moving system $\boldsymbol{\omega} \times \mathbf{r}$ the velocity of the point in the moving system due to this system s rotation and $\mathbf{v}$ the linear velocity of the point in the moving system

Now the acceleration of a point relative to an inertial reference system is given by time differentiation of the previous equation

$$\mathbf{r}_0 = \mathbf{R} + \boldsymbol{\omega} \times \mathbf{r} - \boldsymbol{\omega} \times \mathbf{r} \quad \mathbf{v}$$

As before in terms of components

$$\boldsymbol{\omega} \quad \omega_x \mathbf{i} + \omega_y \mathbf{j} \quad \omega_z \mathbf{k}$$

$$\mathbf{r} \quad x\mathbf{i} + y\mathbf{j} \quad \mathbf{k}$$

and

$$\mathbf{v} - x\mathbf{i} + y\mathbf{j} + \mathbf{k}$$

These can be substituted in the acceleration equation their time derivatives found where necessary the indicated vector multiplications performed and finally terms collected The result is

$$\ddot{\mathbf{r}}_0 \quad \mathbf{R} + \mathbf{a} + 2\boldsymbol{\omega} \times \mathbf{v} + \boldsymbol{\alpha} \times \mathbf{r} - \boldsymbol{\omega} \times (\boldsymbol{\omega} \times \mathbf{r})$$

These terms can be interpreted physically as follows

$\mathbf{r}_0$ is the acceleration of a point *P* relative to the inertial (space fixed) frame

$\mathbf{R}$ is the acceleration of the origin of the moving system relative to the inertial frame

$\mathbf{a}$ is the acceleration of the point *P* relative to the moving system here $\mathbf{a} - x\mathbf{i} + y\mathbf{j} + \mathbf{k}$

$2\boldsymbol{\omega} \times \mathbf{v}$ is defined as the *Coriolis* acceleration of the point *P* $\boldsymbol{\omega}$ is the angular velocity of rotation of the moving axes relative to the fixed or inertial axes and $\mathbf{v}$ is the linear velocity of *P* relative to the moving axes

$\boldsymbol{\alpha} \times \mathbf{r}$ is the *linear* acceleration of the point *P* and depends on the angular acceleration $\boldsymbol{\alpha}$ of the moving axes relative to the fixed axes and on the distance $\mathbf{r}$ of the point *P* from the origin of the moving axes Here $\boldsymbol{\alpha}$ $\boldsymbol{\omega}$

$\boldsymbol{\omega} \times (\boldsymbol{\omega} \times \mathbf{r})$ is defined as the *centripetal* acceleration of the point *P* due to the rotation of the moving axes It is equal in magnitude to $\omega^2 p$ where *p* is the perpendicular distance from the head of the $\mathbf{r}$ vector to the $\boldsymbol{\omega}$ vector it may also be written as $(\mathbf{r}\ \boldsymbol{\omega})\boldsymbol{\omega} - {}^2\mathbf{r}$

As a matter of interest the time rate of change of the acceleration vector can be calculated This leads to the result

$$\dddot{\mathbf{r}}_0 = \ddot{\mathbf{R}} + \mathbf{J} + 3\boldsymbol{\omega} \times \mathbf{a} + 3\boldsymbol{\alpha} \times \mathbf{v} + 3\boldsymbol{\omega} \times (\boldsymbol{\omega} \times \mathbf{v}) + 2\boldsymbol{\alpha} \times (\boldsymbol{\omega} \times \mathbf{r})$$
$$+ \boldsymbol{\omega} \times (\boldsymbol{\alpha} \times \mathbf{r}) + \boldsymbol{\alpha} \times \mathbf{r} + \boldsymbol{\omega} \times [\boldsymbol{\omega} \times (\boldsymbol{\omega} \times \mathbf{r})]$$

where $\mathbf{J}$ is the rate of change of acceleration of *P* in the moving system commonly called the jolt

This equation can also be written as

$$\mathbf{r}_0 \quad \mathbf{R} \quad \mathbf{J} \quad (\mathbf{r}\ \boldsymbol{\omega})\boldsymbol{\alpha} - 3(\boldsymbol{\alpha}\ \boldsymbol{\omega})\mathbf{r} - 3\omega^2\mathbf{v} + (3\mathbf{v}\ \boldsymbol{\omega} + 2\mathbf{r}\ \boldsymbol{\alpha})\boldsymbol{\omega}$$
$$\omega^2(\boldsymbol{\omega} \times \mathbf{r}) + 3(\boldsymbol{\omega} \times \mathbf{a}) + 3(\boldsymbol{\alpha} \times \mathbf{v})$$

The physical interpretation of the terms in this equation is not as simple as in the acceleration equation

# Appendix III

Three graphs are reproduced in this appendix for the determination of

(i) the total acceleration due to gravity plus centrifugal acceleration during rotation and

(ii) the direction of this resultant acceleration with respect to the horizontal plane

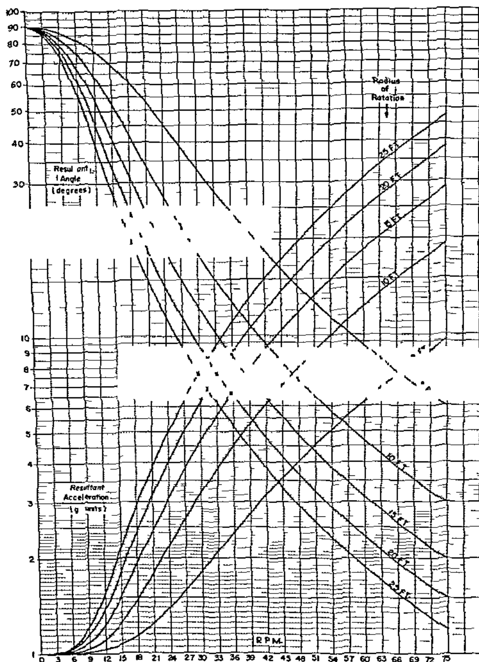



FIG 19 Resultant acceleration in $g$ units (lines converging at the origin) and direction of resultant acceleration with respect to the horizontal plane (lines converging at upper left) plotted against revolutions per minute Curves are drawn for different radii of rotation at intervals of five feet

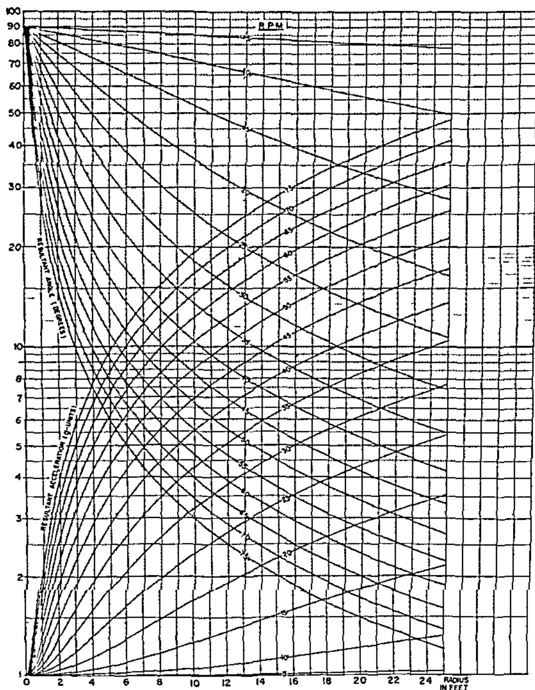



Fig 20 R tant acceleration in $g$ units (lines converging at the origin) and direction of resultant acceleratio respect to the horizontal plane (lines converging at upper left) plotted against radius of r feet Curves for different values of revolutions per minute at intervals of five r p m

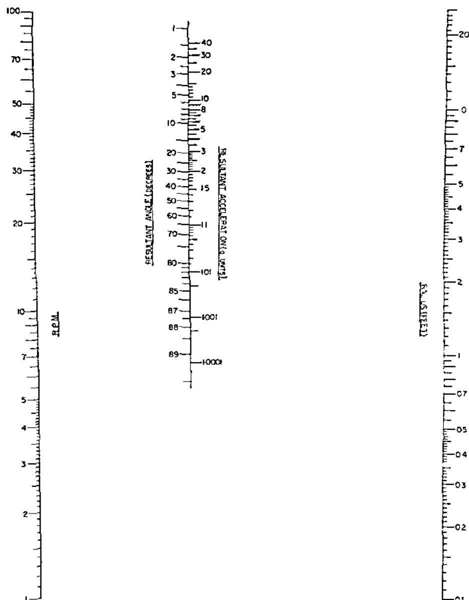



FIG 21 A nomograph from which resultant acceleration and direction can be obtained from a given radius and rate of rotation A straight line drawn from the rate of rotation on the left to the radius of rotation on the right, intersecting the central line, gives the resultant acceleration in *g* units and the angle of this resultant with respect to the horizontal plane

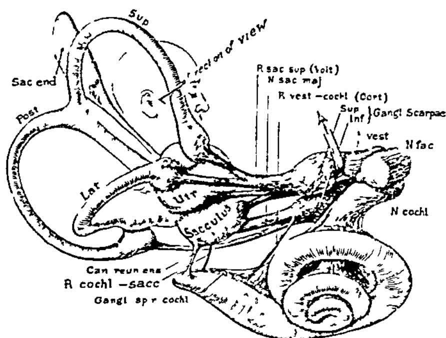



Fig 22 An illustration of the inner ear system on the right side of a human head The two *otoliths* are organs located in the *sacculus* and the *utriculus* labelled Utr (The otolith in the utriculus is thought to be a linear accelerometer some doubt exists as to the function of the other otolith ) The main structures referred to in this report are the three semicircular canals shown on the left of the figure These are labelled Sup —*superior* Post —*posterior* and Lat —*lateral* canals Each canal has an *ampulla* a bulblike enlargement of the canal two of which are shown just where the superior and lateral canals enter the utriculus Within the ampulla is the *cupula* a structure which moves during angular acceleration The canals are filled with a fluid called *endolymph* (Diagram from paper by M Hardy M Anat Rec *59* 403–418 (1934))

TABLE I

# LIST OF SYMBOLS

(Vector symbols which are printed in italics refer to the magnitude of the vector symbols underlined in figures are reproduced in bold type in the text )

| | |
|---|---|
| $\mathbf{a}$ | Linear acceleration vector (p 28) linear acceleration with respect to moving axes system (p 9) |
| $\mathbf{A}$ $\mathbf{A}_x$ $\mathbf{A}_y$ $\mathbf{A}_z$<br>$\mathbf{B}$ $\mathbf{B}_x$ $\mathbf{B}_y$ $\mathbf{B}_z$ | Vectors and their components along the $x$ $y$ and $z$ axes (p 26) |
| $\mathbf{F}$ | *Force vector (p 6)* |
| $\mathbf{i}$ $\mathbf{j}$ $\mathbf{k}$ | Unit vectors along the $x$ $y$ and $z$ axes respectively |
| $\mathbf{i}_0$ $\mathbf{j}_0$ $\mathbf{k}_0$ | Unit vectors in the inertial reference system (p 7) |
| $\mathbf{J}$ | Jolt or time rate of change of acceleration (p 31) |
| $\mathbf{r}$ | Displacement vector (p 6) displacement with respect to moving axes system (p 6) |
| $\mathbf{r}_0$ | Displacement with respect to inertial axes system (p 6) |
| $\mathbf{R}$ | Velocity vector (p 22) displacement of origin of moving axes with respect to inertial axes (p 6) |
| $\mathbf{R}_E$ $\mathbf{R}_N$ | Vector components of $\mathbf{R}$ in east and north directions (p 22) |
| $\mathbf{V}_B$ | Bird's flying velocity (p 23) |
| $\mathbf{V}_W$ | Bird's wind drift velocity (p 23) |
| $\mathbf{V}$ | Linear velocity (p 23) linear velocity with respect to moving axes system (p 7) |
| $\mathbf{W}$ | Angular velocity of subject with respect to moving axes system (p 7) |
| $\boldsymbol{\alpha}$ | Angular acceleration vector (p 28) angular acceleration of moving axes system (p 9) |
| $\boldsymbol{\Omega}$ | Angular velocity of turntable with respect to inertial axes system (p 7) |
| $\boldsymbol{\omega}$ | Angular velocity vector (p 28) angular velocity of moving axes system (p 7) |
| $\theta$ | Smaller angle between the directions of two vectors (p 15) |

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S U P P L E M E N T U M 206

# *THE YOUNG DEAF CHILD:*
# IDENTIFICATION AND MANAGEMENT

PROCEEDINGS OF A CONFERENCE HELD IN
TORONTO, CANADA
ON 8–9 OCTOBER, 1964

ACTA OTO LARYNGOLOGICA KARLAVÄGEN 41, STOCKHOLM O

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 206

# THE YOUNG DEAF CHILD: IDENTIFICATION AND MANAGEMENT

## *PROCEEDINGS OF A CONFERENCE HELD IN* TORONTO, CANADA ON 8–9 OCTOBER, 1964

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STOCKHOLM 1965

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# TABLE OF CONTENTS

# INTRODUCTION

## The Problems

At the University of Toronto two projects directed toward the prevention and the treatment and management of deafness in young children have been in progress for several years One of them has led to the isolation of the virus of rubella This was accomplished under Professor A J Rhodes of the School of Hygiene of the University of Toronto In the other project new electrical techniques are employed to detect cortical evoked responses to sound in young infants, with the hope of ultimately identifying auditory impairments at or soon after birth The second project was a combined effort of the Department of Otolaryngology under Professor P E Ireland and the Department of Physiology under Professor John Scott The principal investigators were Dr Shirley Appleby and Dr W S Goodman Other laboratories began to investigate the same problems at the same time, and the advantages of early exchange of information soon became apparent Also it has been demonstrated, both in England and in the United States, that early and consistent exposure of hard of hearing infants to sound, whether it be amplified sound or merely a parent speaking loudly, aids in the development of normal speech This observation gives importance to the early identification of these partially deaf infants

## The Conference

From these needs and opportunities grew the idea of a conference of investigators and teachers of the young deaf child Its intended purpose, as expressed in the invitation, was "to bring together a small hand picked group of 'experts' to exchange information on and to evaluate methods for coping with the impairment, whether it be by surgery, hearing aids, special education or the exploitation of other sensory channels A leading thought is the hope that fresh ideas for research and perhaps plans for cooperative and even collaborative efforts might develop spontaneously"

Dr Percy E Ireland head of the Department of Otolaryngology of the University of Toronto, took the lead in organizing the conference, acting as host and business manager He was aided and encouraged throughout by Mr E C Fox of Toronto, who explained his interest in the problem in his opening remarks at the conference Dr Hallowell Davis, Director of

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Joan Butcher Helen Hartung B Sue Meyers Adeline Verbarg

## The Meetings

The meetings were held in the Board Room of the School of Applied Science and Engineering of the University of Toronto The luncheons were provided daily for the participants at the Faculty Club by the Faculty of Medicine at the University An official dinner was held at the York Club under the sponsorship of Mr and Mrs E C Fox Senator Joseph A Sullivan acted as Representative of the Board of Governors and the Dean and Associate Dean of the Faculty of Medicine were present

## Organization of the Material

The participants in the conference were selected on the basis of their special experience or of specific contributions that they had made to the topics under discussion The organizer of the program prepared in advance a rather elaborate agenda which is reflected in outline in the table of contents of this volume In the agenda a series of specific questions were formulated in a sequence which was actually followed with only minor rearrangements in the conference itself Most of the participants were requested in advance to be prepared to discuss particular questions or to prepare summary reports on particular topics Other participants indicated certain sections and topics which interested them particularly and also formulated their thoughts in advance In the Table of Contents the names of these primary contributors are given for each chapter

*of hearing in infancy, it is still too early to form an opinion, although many* of the participants were obviously very enthusiastic about the possibilities of this particular method. Others are rightfully skeptical that an instrument cheap enough and simple enough can be developed to satisfy the requirements for really widespread testing, much less for routine screening.

## The Chairman

*During the Conference the Chairman (Davis) frequently violated the* strict rules of parliamentary procedure. He quite properly made numerous *introductory statements and summarized or emphasized important points* in presentations or discussions, and also asked leading questions to provoke further discussion, but on several occasions he abandoned his role as a neutral chairman and became frankly a participant in the discussions. As a participant he presented his personal experiences and advocated his own point of view. In the printed proceedings he has endeavored, now as Editor, to indicate "which hat he was wearing" by designating himself as "Chairman" when he was merely *leading the discussion* and as "*Davis*" when he spoke as an individual participant. As an individual Davis now states that he learned much from the Conference and hopes that readers will find the record of the proceedings both interesting and valuable. As Chairman he thanks the organizers and hosts once more for their hospitality and generosity, and as Editor he thanks the participants for their prompt and whole hearted cooperation in preparing their material for publication.

# I SUMMARY

## Overall Objective

The common overall objective with which the members of the conference are concerned is the alleviation of the handicap to auditory communication which is imposed by a severe auditory impairment that occurs before speech and language have been fully developed

Logically the attack on this problem includes the prevention of early auditory impairment the early and accurate identification of those who suffer the handicap and finally the alleviation of the handicap For alleviation medical and surgical means are used wherever they are appropriate These are supplemented by special care and training with emphasis on the use of amplified sound to enable the child to develop and utilize his remaining potentialities to the full

### 1 Prevention of Deafness

This conference did not review in detail the various possibilities of reducing the incidence of early auditory handicap but it heard with interest and satisfaction an account of the present status of the development of a vaccine against rubella which it is hoped will significantly reduce the number of cases of congenital deafness arising from this source Some attention was also given to the problem of hereditary defects as a basis for congenital deafness and also to the potentialities and limitations of prenatal testing of hearing

### 2 *Identification of the Young Deaf Child*

#### A *Auditory Screening of the Neonate*

The first opportunity to detect severe auditory impairment in young children is during the neonatal period within the first few days after birth Positive evidence of hearing may often be obtained by simple tests based on a startle or arousal response elicited by sudden rather loud sounds ***Methods for routine screening of the newborn infant for auditory impairment have been developed but before widespread programs are initiated the following points must be considered***

1 The incidence of deafness or severe impairment of hearing in the newborn is very low The percentage of children who fail to pass the

screening test in the three most extensive studies ranges from 0.1% to 2.0%. Careful consideration must be given to the value and economy of a screening program whose yield is so low.

2. The validity and the reliability of screening tests of infant hearing are both difficult to establish. Much further research is required. The number of "false positives" and the number of cases missed, which are both rather high, must be considered, the former will cause unfounded anxiety and the latter will give a false sense of security and thus delay later recognition of an auditory impairment. The chance of successful identification of auditory impairment and accurate assessment of its severity becomes progressively greater as the infant grows older.

3. The newborn infant is a very labile organism. He normally sleeps 20 to 22 hours out of each 24 hour period, and on a routine basis it may be difficult to catch him in an optimal state for testing. The best state is asleep, but he may difficult to arouse sufficiently from deep sleep. He is hyperactive and tense when hungry or cold.

4. Certain conditions which can affect the auditory system, such as anoxic brain damage and particularly hyperbilirubinemia, may not show their effects the first day or two. Hyperbilirubinemia in particular may not cause symptoms before the 4th or 5th day or even later.

5. After they leave the newborn nursery only a small percentage of infants in most communities can be brought together for screening purposes. The maternity hospital thus offers unique opportunity for screening. Against this must be weighed *a*) the economy of a neonatal screening program, *b*) the question of whether effective remedial measures, special training or management are available, *c*) whether a delay in initiating the remedial measures or the special training and management is of critical importance, and if so *d*) how long a delay is critical.

Opinions differ on the matter of how long a delay is allowable in the case of partial auditory impairment. There is no clear evidence that a delay of 6 months is critical from the point of view of development of language. Most participants in the conference agree that a year is probably critical, and many feel that the sooner auditory experience and training can be provided the better.

6. The possibility of injury to hearing as the result of misplaced and unnecessary use of amplified sound or of other loud and continued noise cannot be overlooked. Certainly the use of amplified sound for infants during the early weeks of life must be instituted with due caution. Indications of pain or discomfort are important guides, and an infant 6 months old can register them more clearly than a neonate.

## B. *A Positive Program*

*It is not necessary to wait for the methods of routine auditory screening at birth to be perfected and validated or for its economic feasibility to be*

demonstrated An effective program for the early identification of children likely to have problems in communication can and should be instituted immediately Such a program might be developed in two steps

1 *A high risk register* should be instituted containing the names of babies in whom, for one reason or another, the risk of an auditory handicap is substantially higher than it is in the general population The indications for placing a baby in a high risk register are given in detail below

Babies at risk should be followed closely from the point of view of development of normal auditory behavior, and if deviations are suspected definitive testing should be carried out The children at risk should be seen fairly frequently during the first two years, say at 0, 3, 6, 9, 12, 18 and 24 months

2 *All children attending well baby clinics* or coming to pediatricians' offices might be screened during the latter part of their first year by a simple but well planned test or else by a questionnaire pertaining to auditory behavior, such as the communicative evaluation chart developed by Anderson, *et al* * Children who do not yield satisfactory responses on two test occasions or concerning whom doubt is raised by the questionnaire would then be referred for more definitive testing

The success of such a program will depend on the education of physicians, public health personnel and, above all parents with respect to normal expectations for the development of hearing and language and the possibility of auditory impairment Among physicians it is particularly important to alert both obstetricians and pediatricians

It would be ideal if impairment were detected and confirmed by 6 months of age but a more practical time to try to identify the deaf child and institute appropriate special care and training is during the second half of the first year This is a compromise between reliable detection of impairment and the earliest possible start on special auditory training

## High-risk Register for the Better Identification of Children with Communication Problems

I *Antenatal*

a) Positive family history of deafness
b) Familial biochemical abnormality associated with deafness
c) Blood incompatibility (Rh factor)
d) Virus infection during early pregnancy
e) Bleeding, especially during the first trimester
f) Drugs, notably any of the mycin group or quinine

* *Communicative Evaluation Chart from Infancy to Five Years* Compiled by Ruth M Anderson and Madeline Miles Speech Department and Patricia A Matheny Audiology Department Children's Hospital Denver Colorado Copyright 1963 by Ruth M Anderson Madeline Miles and Patricia A Matheny

II *Complications of Labor*

a) Premature delivery
b) Fetal distress due to maternal shock, etc.
c) Prolonged or precipitate labor
d) Difficult delivery—traction on neck or birth injury

III *Neonatal Difficulty*

a) Apnea or cyanosis
b) Cerebral birth injury
c) Jaundice—hyperbilirubinemia (15 mg/cc and above).
d) Multiple anomalies—from whatever cause
e) Possible iatrogenic trauma, as noise of an incubator, drugs (notably streptomycin and kanamycin), etc

IV *Factors in Early Childhood*

a) Infections, such as meningitis and measles
b) Chronic respiratory infection and/or allergy
c) Injuries
d) Hypothyroidism
e) Abnormality of external ear.

V *Possible Social Factors*

a) Maternal mental retardation
b) Socio-cultural deprivation—poor child care, etc
c) Emotional problems

Items I, II and III are the particular concern of the obstetrician, items III, IV and V of the pediatrician

## C. *Types of Test*

Several types of auditory test have been developed They rest upon different classes of physiological or behavioral response Some are more suitable than others for very young children and they vary also in the definiteness of the information which they yield Many of the tests are quite satisfactory within established limitations, but further research is required We need to understand more fully the expectations in the normal development of children and we also need to improve our techniques, both for screening and for definitive diagnostic testing In particular, further analysis in terms of simple reflexes, thalamic and cortical sensory systems, the [illegible] second signal system", and conditioned reflexes is needed

T[illegible]us types of test discussed specifically in the conference are listed below [illegible]ly in the order of their applicability by age

1. Early screening tests may be based either on simple noise makers or on specific acoustic signals, electronically generated. These tests yield qualitative rather than quantitative information. The criteria include the startle response (Moro reflex) or other general muscular movements, eye blinks, changes of respiration or heart rate, a pause in sucking if the infant is feeding, or indications of awakening from sleep. There are also responses to quiet sounds and, at 4 months of age, a characteristic pre-orientation reaction.

2. A second group of tests suitable, after the sixth month, depends on an *orientation reaction, i.e. turning of the eyes and head to locate the source* of sound.

3. Another type of test is based on electrical phenomena, notably the electroencephalogram (EEG) and the so-called "electro-dermal" (ED) or "skin galvanic" response. Much more elaborate equipment is required for such tests but the results may be more quantitative and definitive than for the first two classes. However, electrodermal testing is not satisfactory for infants. A recent modification of EEG audiometry using averaged evoked responses gives great promise both in older children and in infants even down to the neonatal period, but the technique is still under development. A simplified and less expensive set of equipment must be devised before the method will be available for widespread use.

4. The final class of test merges into classical pure-tone audiometry. Classical audiometry can be adapted for young children, sometimes down to even the two-year age level by such devices as the familiar "peep show" and "play audiometry." The latter in particular is now in general use in many clinics. The reliability of pure-tone audiometry increases rapidly with the age of the child.

## D. *Differential Diagnosis*

Other disorders or handicaps may be confused with auditory impairment or may combine with hearing loss to make more severe the difficulty of communication, the acquisition of speech, and the learning of language. The key to correct diagnosis and proper handling is the correct assessment of the degree of peripheral auditory impairment, i.e. to determine whether the child fails to respond to acoustic signals because auditory nerve impulses never reach the brain or because something interferes with the will or with the ability to respond, or both. If the acoustic signal is speech it is less effective for children who have not learned the meaning of sounds and words. Particularly important as confusing conditions are mental retardation, autism, and other psychiatric conditions, including schizophrenia. More obvious but equally important is cerebral palsy, where the auditory impairment may not be adequately recognized because of the more evident disturbances in the motor system. A much discussed but perhaps over-emphasized condition is "congenital aphasia" or "central auditory imperception" or "central dysacusis" in which the brain rather than (or in

addition to) the ears or the organs of speech is at fault. The auditory signals, although transmitted to the brain as nerve impulses, do not acquire meaning.

An important differential diagnosis is to distinguish between central dysacusis and an emotional pattern of behavior based upon an unrecognized peripheral auditory impairment. Speech sounds may be heard but imperfectly, with consequent failure to acquire language. Enough may be heard, however, perhaps in the very low frequencies, to indicate an awareness of sound so that the auditory defect is not obvious. The understandable failure of parents to recognize the nature of the difficulty and the resulting difficulties and behavior problems are easily understood in retrospect. The difficulty of acquiring language late after such a bad start emphasizes the importance of correct and early identification of the deaf child and the institution of proper special training and management.

## MANAGEMENT

### *Medical and Surgical Treatment*

Medicine and surgery between them can alleviate or even eliminate conductive hearing loss. One of the great benefits of systematic screening of all children for auditory defects near the end of the first year and again upon entering school (with additional tests of hearing for any child who *does not learn to talk spontaneously by the end of the second year*) would be the detection of curable conductive impairment. Some conductive impairments are due to congenital malformations. These are usually obvious and often can be helped by surgery.

The commonest cause of conductive impairment is otitis media (infection of the middle ear). Prompt and proper treatment of a discharging ear may prevent permanent hearing loss. Milder infection may produce a less obvious mucoid or a serous otitis media that can escape attention completely yet interfere seriously with the learning of language. This condition is completely amenable to treatment. The commonest basis for otitis media is chronic or repeated infections of the upper respiratory tract.

### *Parental Attitudes and Skills*

For the successful management of children with auditory impairments that are sensory-neural or central and therefore cannot be assisted by medical or surgical treatment, *the most important single factor by far is loving, intelligent and consistent parental care.* The proper slogan is "bathe the child in sound." The human voice speaking loudly and clearly close [illegible] louder than we often realize and can usually reach any really [illegible] of hearing. Such a residue is usually present, particularly

in the lower frequencies The child must be made aware of sound, through his auditory system if possible, to give the best possible chance for normal development and organization of the parts of the central nervous system that are involved in the understanding and production of speech The period from six months to two years is particularly important, and during *this period the best possible sources of sound are the voices of its parents,* speaking loudly, close to the ear The best *form* of sounds is their spoken words The concepts and the techniques are relatively simple, but much guidance and training in techniques is often needed by parents Appropriate counseling, encouragement and guidance, particularly in the early months, may spell the difference between success and failure for the child s development of language Some children who are severely deaf may still fail to develop language normally, but the effort should always be made

## *Amplified Sound*

Amplified sound provided by a hearing aid may greatly assist the development of speech and language in young children with auditory impairments that are not too severe Normal development of speech requires that the child hear his own voice as well as the voices of others It is now feasible to provide children as young as six months with hearing aids, thanks to the development of individually fitted plastic insert earmolds New molds must be provided periodically as the child grows, but the child becomes accustomed both to the mold and to the hearing aid, which may be worn during many of the child's waking hours

The importance of a hearing aid depends upon the severity of the hearing loss An aid is particularly helpful if the hearing loss is severe but not total Ideally an instrument should be provided as soon after six months as the diagnosis of a hearing loss that is not amenable to medical or surgical treatment is established by definitive test The definitive tests, the beginning of special training, and the use of amplified sound should certainly be initiated before the end of the second year By that time, and perhaps even earlier, the opportunity to take advantage of natural processes of development is reduced, the discrimination of sound patterns, the appreciation of their significance for communication, and the control of the infant s own voice in imitation become less easy and spontaneous If the opportunity is missed at the "natural" age it becomes progressively more and more difficult for the child to develop speech and language later, even *with the best of special instruction*

Two important precautions must be observed with hearing aids

1 *The diagnosis of peripheral hearing loss must be clearly established* If the failure to respond to sound is due to a central defect, whether organic or non-organic, there is danger of overloading and possibly injuring a normal or nearly normal ear The reaction of the child, particularly in-

dications of discomfort when the hearing aid is turned on, must be carefully observed and respected

2 *For children with fragmentary hearing for low frequencies only, hearing aids with extended low-frequency range are more effective* than aids with the usual response characteristic that favors high tones and attenuates the low Conventional instruments are suitable for children who can still hear high tones, but for those who are severely impaired, *with only a residue of low-tone hearing remaining, a hearing aid that does not amplify low-frequencies adequately is not as good as the unaided voice speaking loudly* close to the ear

Amplified sound will not restore sensory cells or nerve cells that have failed to develop normally or have been destroyed by toxins, drugs, or lack of blood supply It will, however, promote and give the best opportunity for the natural and most effective organization of such auditory faculties as have been spared

The requirement for hearing aids for young deaf children is now clearly established Consideration should now be given to the detailed specifications, the design, and the manufacture of hearing aids intended especially for this use The question of adequate bandwidth is particularly important

### *Special Education and Remedial Education,*
### *particularly with respect to language, in the pre-school and early school years*

Special education and intelligent well-planned home care, with or without additional formal instruction of parent, child or both, is much easier and more effective than remedial education for the child at a later period Early special education, preferably assisted by amplified sound, also greatly reduces the emotional stresses and behavior problems that all too frequently complicate a primary auditory impairment The special education that is needed is directed particularly to the development of speech and language The exact form of such instruction and the expectations for its success depend critically on just how much of the peripheral auditory system remains intact and functional and what the child's hearing threshold levels are at each frequency

Many misunderstandings have arisen among workers, particularly workers in different countries, from differences in terminology The word "deaf" is used by some to mean any degree of auditory impairment that is severe enough to cause any real handicap Others use it only to indicate total loss of true hearing Still others use it to designate all children who hear too poorly to develop normal speech spontaneously Others may speak of deafness as being 'cured' when a child is taught to understand amplified speech with the emphasis on the lack of understanding rather than on th[illegible]ted auditory thresholds For these reasons the word "deaf" has b[illegible]led in the present summary

Another misunderstanding arises from the difference in the calibration of pure-tone audiometers in America as opposed to Europe and Great Britain The difference is about 10 decibels A hearing threshold level for speech of 60 dB American corresponds to about 70 dB British (or International)

Almost all children, even those with no sensory cells or auditory nerve fibers, will give a positive response to the loudest low-frequency signals from a modern pure-tone audiometer Even totally deaf children usually respond to the tactile or vibratory stimulation, either in the external or in the middle ear This sensation, although it is not true hearing, may be useful to them and should be utilized to the full, if only to help them improve the quality of their own speech, but it is insufficient for adequate analysis of the complex signals of spoken language A remnant of true hearing, particularly if it extends as high as 2000 cps, makes a critical difference in the prognosis A child with such a remnant can usually learn to understand speech fairly well with the help of a good hearing aid

The more hearing that remains, all other factors being equal, the easier is the task of teaching and the greater the expected level of ultimate performance The great success of auditory training and other special instruction with children who are simply hard of hearing must not either raise false hopes for the children who are truly deaf or condemn methods of teaching that fail to give them perfectly modulated voices or the ability to understand speech by the sense of hearing alone More accurate assessment of the hearing of children at the earliest possible age will help us to provide for each child the type of special care and instruction that is needed for his particular type and degree of impairment

Fortunately the number of children who are simply very hard of hearing and for whom the prospects are relatively bright is considerably larger than the number of less fortunate children who are truly deaf and who can only feel but never hear sound The totally deaf, who need to depend on lipreading for speech reception, can usually be taught to speak The degree of success in these cases is largely dependent on the age at which training is begun and the methods used Resort to the language of signs and the *manual alphabet is much less often necessary when an early beginning* is made

# II. PREVENTION OF DEAFNESS IN VERY YOUNG CHILDREN

## A. Opening Address — Mr. E. C. Fox, Toronto

Mr Chairman, Dr Ireland, Dean Hamilton, and Dean MacDonald My wife was asked by two or three people how we came to get into this thing This is how I had a grandson who was my first grandson and who was a rubella baby with a bad heart, bad ears and bad eyes I hope you will greet him tonight in person at the dinner, because I'm rather proud that he is Exhibit A" of what we are advocating here He was brought up by his own grandfather shouting at him, so that whatever residue of sound there was, he got Dr Silverman, who was in Toronto shortly after we found that the boy was deaf, saw him, and the one sentence he said that I remember very clearly was "Bathe the child in sound That struck me as awfully good commonsense, and having a rather strident voice myself (my mother was very deaf), I bathed the child in sound and spent all my spare time with him He was living in our house as his father was overseas in the War

The boy acquired the power of natural speech very fortunately Dr Wishart, who was his ear doctor at that time, said that he thought he could acquire natural speech if his grandfather and mother would do their part And from that I realized that, no matter what deliberations we may have here today, or what conclusions you may come to regarding clinical treatment or methods of teaching we cannot escape the duty that lies at home with deaf children

Now I realize that most of the hard-of-hearing children and deaf children come from poor homes, where the mother is busy preparing the meals and looking after other children, and the father is busy earning a living and when he gets home in the evening his energy is exhausted and he has no reserves left to give to what will turn out to be a backward child And so the backward child gets neglected

Now, that s one of the things that I'm sure the layman can speak to you about I can't talk about any of the technical matters, but I can say this to you you must never cease to stress the home obligations and, to the extent that the parents, after being warned, do neglect this, the result unfortunately registers on the poor little child as it grows up

The occurrence of deafness I don't think ever bothered me very much

My grandfather never heard my voice He was totally deaf He went deaf in his twenties for catarrhal reasons they said I expect that is perfect nonsense My mother also went deaf when she was 22 and I wasn t born until she was 30 so I always had to shout very very loudly at my mother and developed a strident voice Then deafness missed a couple of genera tions It apparently wasn t passed on but this little boy got caught by rubella

Now I want to talk for a moment about rubella Two projects were launched at the same time this Identification and Management project and the Rubella project Within a year they had the virus at the University of Toronto and also the Walter Reed Institute in Washington made a similar announcement and last evening Dr Janet Hardy told me that a third institute also has it so that there are three institutions now trying to develop the vaccine that will be distributed I hope throughout the world May God bless the winner of the race because I hope they have some regard for the time element

I don t know whether anybody today will be able to make an announce ment as to when this vaccine will be out I had hoped that it might be in 64 but possibly one of the institutions in the United States may have it out in 65

Now how big a residue is left? There must still be a very big residue of congenital deafness but how big we don t know

Now back to my grandson The little fellow put me into a panic when it was discovered during an interview with the doctor that he was hard of hearing And even then and that was in 43 the methods of proving the amount of deafness and the degree of impairment must have been pretty accurate because Dr Wishart at that time guessed a 70 decibel loss (Amer ) and this is just the loss that has stayed with the boy He assured me that it would never get any better or worse and that if the parents and grandparents did their duty he would have the power of normal speech Now he does have the power of normal speech thank God Anyway I live in dread whenever I see hard of hearing children and think of the awful handicap that they have to face My boy has faced it don t make any mistake about that He will never be quite normal but to lessen the incidence of disadvantage and embarrassment that is very worthwhile aim

I thought I was doing good work in trying to supply hearing aids to people who couldn t afford them until I suddenly woke up and realized that this was entirely inadequate I said to myself Let s get to the *source* of this thing

You may not all agree with Dr Griffiths I know that she has aroused controversy from time to time but it was Dr Griffiths of Los Angeles who gave me my two ideas that led to these two projects I said to her one morn ing Has nobody tried to isolate the virus of rubella? She said Well I think Mr Fox you will find that they have tried they must have but nobody seems to have done it And I said Is it worthwhile trying again?

She said, "I think it is", and that led to interviews with Dean MacFarlane at the University of Toronto, and ultimately, to the project of the isolation of that virus

The other thing I asked Dr Griffiths about was the development of an instrument which would detect impaired hearing as close to the natal day as possible, and which would one day measure the amount of hearing Of course, in my simple layman's idea, I thought that was quite simple and it could be done, but I very soon found that it may not be quite so easy

I don't care whether what we have developed here at the University of Toronto, and is to be presented by Dr Ireland or one of his colleagues, is the one that is adopted or not, so long as we have stimulated something Dr Griffiths stimulated my mind to do something, and I was able to help the Toronto University in the only way I could help I am glad that I have done it My heart's in this thing, and I hope that today and tomorrow we will give a push by which we will improve the medical and the social status of these young children, so that they won't have to face such handicaps of life as they otherwise would So, God bless you, and now let me disappear out of the picture

CHAIRMAN In his introductory remarks the sponsor of this conference pointed out that the attack on the problem of deafness in very young children is really a three-pronged attack first, prevention of congenital or acquired deafness as far as possible, second, early and accurate identification of all deaf children, and thirdly, the most efficient management and training of those whose deafness cannot be alleviated by medicine or surgery The discussions of this conference will deal with the second and third of these points, namely the identification and the management of the very young deaf child, but as an introduction we shall hear a brief report of the status of current work on the identification of the virus of rubella and the possibility of eliminating this particular cause of congenital deafness by means of a vaccine Such a review is particularly appropriate because the University of Toronto is one of the institutions that recently identified the virus of rubella

## B. Prevention of Rubella

J HARDY The matter of rubella infection in pregnancy has been of particular interest in the "Collaborative Perinatal Study" in which I am engaged and which I shall outline shortly for you In this study we have had an unusual opportunity to identify the infection and also to follow the children of mothers who have had it

The virus of rubella was isolated about two years ago at several places almost simultaneously Very close on the heels of this announcement, Dr

John Sever at the National Institute of Health, who was one of those who had isolated the virus, also developed serologic tests which could be applied to patients in the collaborative projects This test was applied to some 20 000 women in the projects and he found that about 80% of these women already had antibodies to rubella at the time they became pregnant For these women rubella was not a problem However, we can at the same time conclude that there are about 20% of young women who do not have these antibodies The percentage of those without the antibodies is higher among the younger mothers than among the older ones, and for all of them without the antibodies rubella is a danger

As soon as the virus had been isolated, the next step was to make a satisfactory vaccine that could be used to immunize young girls so that when they became pregnant they would not be vulnerable to the rubella infection This has not proved to be a very simple or easy job There are other virus infections in monkeys such as the TPLL virus which have interfered to some extent and confounded the problem However, progress is being made and I think we can look forward with confidence to having a rubella vaccine generally available within the next two years

I would like to add that during the early spring of 1964 we had a serious outbreak of rubella along the eastern seaboard of the United States It was quite extensive and the disease was unusually severe Many young adults were stricken and for the first time it was possible to be sure that this really was rubella, because the virus was isolated and identified The babies from women who had rubella or who were exposed to it during this period are just beginning to appear in the nurseries We had one baby in the last month whose mother was exposed but who had had no rash The baby was born with cataracts and congenital anomalies and microcephaly This triad is characteristic of rubella We do not yet know whether this baby will have a hearing defect

Other observers have also found that exposure to rubella, presumably followed by a "subclinical" infection without rash, can indeed result in anomalous infants We now have in our collaborative perinatal study 125 mothers who were exposed but who had no rash We have the serological studies on them and in the next few months their babies will arrive We are planning to follow these babies carefully to ascertain the possible bad effects from such exposure to and possible subclinical infection by the disease

In the past, rubella has been a difficult disease to identify with certainty Infection with the coxsackie virus and probably others as well produces rashes very similar to the rash of rubella With the serological studies that are being routinely done on our mothers in the collaborative projects we should be in a much better position to assess the importance of the rubella virus

Another interesting and important fact about rubella virus is that it may remain in the foetus for many weeks We have studied the foetuses of

women who had therapeutic abortions because of actual infection with rubella in the first weeks of pregnancy and we have been able to isolate the virus from a large percentage of these foetuses some of them as long as 16 or 17 weeks after the mother had her clinical rubella Possibly this virus may continue to damage the nervous system through loss of nerve cells even after the mothers have recovered from their clinical infections

CHAIRMAN Thank you Dr Hardy It was a most illuminating report of progress and your opportunity to take full advantage of one of nature's experiments the rubella epidemic of 1964 should really yield a rich harvest of additional knowledge Through the efforts of those who isolated the virus and developed the serological tests and the vaccines and of those who now study the effects with definite knowledge of the etiology we may look forward to the control and elimination of this particular variety of congenital deafness We can hope that in the future still other causes may be identified and eliminated one by one but unfortunately we must admit that in far too many cases the cause of the congenital deafness is still unknown One broad class however, has been clearly identified namely hereditary deafness which depends upon chromosomal (genetic) defects Dr Wedenberg of Stockholm will illustrate this problem by a description of a particular variety of hereditary deafness that he has had opportunity to study

## C HEREDITARY DEAFNESS

WEDENBERG There is one form of hereditary hearing loss which is particularly deceptive These children are born with normal hearing but they lose their hearing during the first years of life I have followed several of them very carefully and have made records of them and their audiograms When I examine the parents I find that most of them have either great clinical defects of hearing or subclinical defects of hearing i e measurable abnormalities which do not constitute a serious handicap The condition depends upon genetic factors which lead to degeneration within the cochlea I have looked up one particular family line back to the 16th century In this family the children are born with normal hearing but many of them lose that hearing by the age of 21 In the present generation two children are 13 and 7 years of age and they have already lost a great deal of their hearing Their father was born with normal hearing but is now totally deaf

CHAIRMAN There doesn't seem to be very much that can be done about this type of hereditary defect except perhaps through the approach of eugenics The situation is deceptive because the child's hearing would appear normal by any screening test in the early years

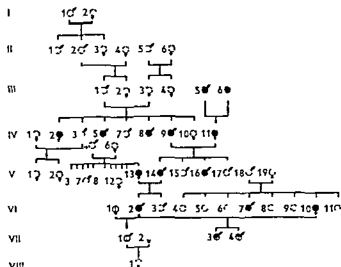


FIG II 1 Pedigree of eight generations in which the affected individuals (half filled or full circles) became gradually hard of hearing or deaf

## D. PRENATAL TESTS OF HEARING

CHAIRMAN Dr Wedenberg has long been interested in the problem of congenital deafness, whether it arises from genetic defect or from virus infections such as rubella He reasoned that if it were possible to test the hearing of the foetus long enough before birth the result of such a test might contribute strongly to the decision whether a therapeutic abortion might be justified or not In other words if a mother were known to have

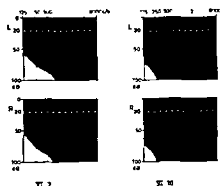


Fig II 2

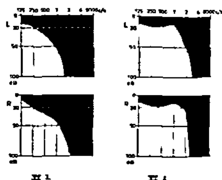


Fig II 3

FIG II 2 Audiograms of the totally deaf father and mother of the 6th generation (VI 2 and VI 10)

FIG II 3 Audiograms of the two hard-of hearing sons (VII 3 and VII 4) taken at the age of 13 and 7 years respectively They showed a marked deterioration in comparison with earlier audiograms

women who had therapeutic abortions because of actual infection with rubella in the first weeks of pregnancy and we have been able to isolate the virus from a large percentage of these foetuses, some of them as long as 16 or 17 weeks after the mother had her clinical rubella Possibly this virus may continue to damage the nervous system through loss of nerve cells even after the mothers have recovered from their clinical infections

CHAIRMAN Thank you, Dr Hardy. It was a most illuminating report of progress, and your opportunity to take full advantage of one of nature's experiments the rubella epidemic of 1964, should really yield a rich harvest of additional knowledge Through the efforts of those who isolated the virus and developed the serological tests and the vaccines and of those who now study the effects with definite knowledge of the etiology, we may look forward to the control and elimination of this particular variety of congenital deafness. We can hope that in the future still other causes may be identified and eliminated one by one, but unfortunately we must admit that in far too many cases the cause of the congenital deafness is still unknown. One broad class, however, has been clearly identified, namely hereditary deafness, which depends upon chromosomal (genetic) defects Dr Wedenberg of Stockholm will illustrate this problem by a description of a particular variety of hereditary deafness that he has had opportunity to study

## C. HEREDITARY DEAFNESS

WEDENBERG There is one form of hereditary hearing loss which is particularly deceptive These children are born with normal hearing but they lose their hearing during the first years of life I have followed several of them very carefully and have made records of them and their audiograms When I examine the parents I find that most of them have either great clinical defects of hearing or subclinical defects of hearing, i e measurable abnormalities which do not constitute a serious handicap The condition depends upon genetic factors which lead to degeneration within the cochlea I have looked up one particular family line back to the 16th century In this family the children are born with normal hearing but many of them lose that hearing by the age of 21. In the present generation two children are 13 and 7 years of age and they have already lost a great deal of their hearing Their father was born with normal hearing but is now totally deaf

CHAIRMAN There doesn't seem to be very much that can be done about this type of hereditary defect except perhaps through the approach of eugenics The situation is deceptive because the child's hearing would appear normal by any screening test in the early years

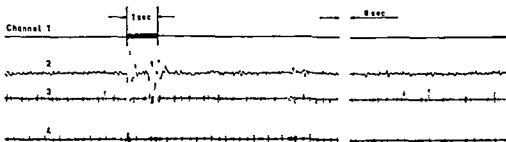



FIG II 7 Recording of the reaction of the foetus to a tone of 3000 Hz, 110 db *SPL* for 1 sec Channel 1 the test tone channels 2–4, three different bands Case 3, 2nd series A general muscular reflex indicated in channel 2

stimulation of the foetus we found it desirable to use a stimulus tone in the higher frequency range, and we chose 3000 Hz, since the physical measurements showed that this was the highest frequency at which acceptable stimulation conditions could be obtained The arrangements for calibrating of the system are shown in Fig 4 The whole surface of the vibrator is placed in close contact with the body of the subject and a *microphone is inserted in the uterus*

By means of the bars A, B and C the distance between the microphone and the vibrator has been calculated For a distance of about 10 cm the sound pressure level at 3000 Hz was 110 ±6 db (Br ), limits obtained on four subjects A typical frequency response curve is shown in Fig 5 Mothers with 2 7 weeks to term were selected for the investigation The mother was placed on a bed with the head lowered to minimize circulatory disturbance due to the supine position In spite of this, fainting attacks occurred in some cases The position of the head of the foetus was determined and the decided position of the vibrator was marked on the skin The microphone of a phono-cardiograph was placed where the beats of the foetal heart were best heard (Fig 6) The subject was asked to relax

After a rest of 5–10 min the control measurements were started, these included a recording of the beats of the foetal heart without stimulation A sharp clap behind the mother's head, the application of the vibrator and a broad band masking noise applied to both ears of the mother did not interfere with the reaction of the foetus

By means of a timer unit, T in Fig 6, a pure tone of 3000 Hz was presented for 1 sec at a sound pressure level of 110 db (Br ) At a frequency of 3000 Hz a vibratory sensation at the skin of an adult is excluded and this can also be assumed for the foetus The pulse rate of the foetus was recorded with the phono-cardiograph Under the presumption that other reactions to the stimulus can be disregarded, a change in the pulse rate can be considered as a reaction of the auditory system

In the phono-cardiogram reproduced in Fig 7 movements of the foetus are distinctly depicted, these correspond to direct observations In Fig 8

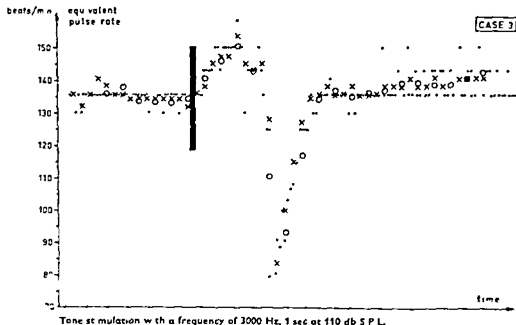



FIG II 8 The reaction of total pulse rate to the tone, indicated by the vertical thick line plotted against time; •, equivalent pulse rate, beats/min, every beat indicated, x, equivalent pulse rate, beats/min, integrated over three beats, O, equivalent pulse rate, beats/min integrated over six beats Case 3, 2nd series

the same recording is represented with different integrating periods Integration over six beats was chosen.

Twelve foetuses were examined Four or five tests were performed on all From 45 tests on 10 foetuses 35 showed a highly significant deviation $P < 0\ 001$, five tests $0\ 001 < P < 0\ 01$, two tests $0\ 01 < P < 0\ 05$ and only three non-significant deviations of the increasing pulse rate Postnatal tests showed that the hearing of all the subjects was normal over the frequency range 500–4000 Hz

In later experiments we tried to establish at what point in the uterine development a reaction by the foetus to sound can first be recorded Histologic research has shown that the cochlea and the sensory end-organs have reached their normal development at the 24th foetal week and therefore from this time a reaction to sound *might* be possible On a series of 15 women the reaction of the foetus was examined to frequencies 500, 1000, 2000 and 3000 Hz at an intensity of 110 $\pm 6$ dB (Br ), beginning at the 22nd week While the results are not yet clear, it is certain that there was no reaction to sound of any of the foetuses in the 22nd week. Definite reactions were recorded in the 26th week and they became increasingly distinct with foetal age Postnatal hearing tests have shown the hearing of these children to be normal

CHAIRMAN Thank you Dr Wedenberg You have demonstrated that reactions of the foetus to sound delivered through the mother s abdominal wall can regularly be obtained as early as the 26th week of pregnancy but not earlier The earliest possible date for such a reaction is about the 24th week when innervation of the cochlea begins The practical application of these finds is limited in Sweden by legal restriction

WALTER Have you any evidence as to whether the reactions might depend upon vestibular rather than cochlear stimulation? In the adult we know that there are many responses to apparent auditory stimulation which are actually mediated through the vestibule If this is possible might there not be an error in this method of appraisal? A normal response might be mediated through a vestibule which is in fact normal while the cochlea might have failed to develop

WEDENBERG We have chosen the test frequency 3000 Hz to be sure of no tactile stimulation We have no direct evidence of a cochlear reaction and of course a vestibular stimulation might be possible and for higher levels is highly probable For the levels used in our experiments we think however that it is a cochlear reaction but we have stated in the report the *reaction* not hearing by the human foetus

RØJSKJAER Do you think there is any possibility of injury by sound to the cochlea or other structures in the petrous bone?

WEDENBERG I have tested all of the infants after birth and they all have had perfectly normal hearing The intensity of the stimulus that actually reaches the foetus should be well within safe limits

DAVIS I think we should recognize that the stimulation of the cochlea of the foetus in this situation is by bone or perhaps rather by fluid conduction There is no air in the middle ear cavity to allow reciprocal or differential movement of the stapes and the round window The measurements of sound level made by introducing a microphone into the uterus after delivery must be regarded as only very approximate unless the microphone were in fact designed for measuring underwater sound I will ask Dr Wedenberg first whether he agrees that stimulation is essentially by bone conduction and second whether he did in fact use a microphone designed for measurements of underwater sound Actually there does not seem to me to be any real probability that dangerous sound pressures are delivered to the foetus in this situation

WEDENBERG For the calibration tests of the levels inside the uterus we made a special technical calibration in a water tank In this way the type of microphone is of minor interest The air volume in front of the

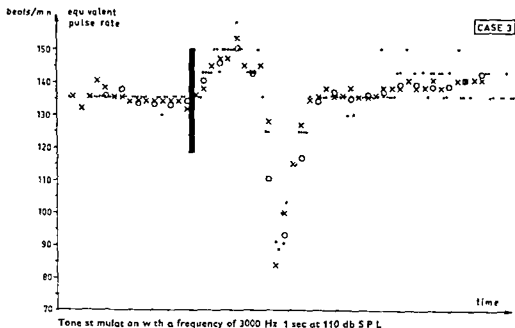



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# III IDENTIFICATION

## A Early Post Natal Tests

Chairman The method of prenatal testing of hearing described by Dr Wedenberg is obviously not suitable for a routine screening test to be applied to the general population It might in principle be applied to cases where there is strong reason to think that a hearing defect might be present but practically speaking the earliest moment at which we might hope to identify the children with auditory defects is in the delivery room or in the nursery within the first hours after birth The concept of such perinatal screening for auditory defects is a familiar one and its potentialities have now been explored thoroughly and completely by several investigators including Dr Janet Hardy of Johns Hopkins University

Before calling on Dr Hardy in introducing the subject of identification of the very young deaf child I wish to point out that we shall encounter two opposing considerations One of these is that the actual percentage of children with significant impairments of hearing is very small Therefore any screening test that is to be applied to every child in the nursery or at some later period must be quick simple and inexpensive to justify taking time to apply it to every child Another consideration is that it is poor strategy to apply a screening test until the infant or child is old enough to make the test reliable Otherwise we shall have too many false positives and shall also miss too many cases These considerations argue against very early testing On the other hand we shall hear tomorrow that there is good reason to believe that it is important to identify deaf children quite early in order to begin special training and provide them with sound stimuli during the period of natural development of the auditory system and the related development of the voice speech and language These considerations argue as our sponsor pointed out in the introductory remarks for the earliest possible identification of the deaf infant Dr Hardy s study is one of special importance because it will give us some definite evidence by which to evaluate the effectiveness and consequently the desirability of testing hearing during the neonatal period Tomorrow when we consider the problems of management we will ask the opposite question namely at just what age does it become really important to begin auditory training? Can we spare a few months without a significant loss of auditory development in the long run?

Dr Hardy, will you first tell us a little about the so-called "Collaborative *Perinatal Project" and then the results of your testing of hearing in the* newborn?

## *Collaborative Perinatal Project*

J HARDY In 1958 the National Institute of Neurological Diseases and Blindness organized a project known as the "Collaborative Project for the Study of Cerebral Palsy, Mental Retardation and other Neurologic and Sensory Disorders in Children" This descriptive but cumbersome designation has now been replaced by the more inclusive and general title of "Collaborative Perinatal Project"

Fourteen institutions joined with the NINDB in this project It is conceived as a longitudinal multidisciplinary study of approximately 57,000 pregnancies The basic problem is really that of pregnancy wastage in the broad sense, extending from the extreme of early spontaneous abortion to the poor performance in school of a child with minimal brain damage The number of pregnancies required in this study is large to allow proper statistical analysis and therefore the collaboration of many institutions was essential

At the Johns Hopkins University the women who register in the Women's Clinic of the hospital are included in the study This is a ward population not including any private patients, and approximately 70% of the women are negro The last baby will be accepted into the study about August, 1965 By that time we will have a population of approximately 4500 women who became pregnant and between 4000 and 4200 living children Our follow-up studies so far are quite successful in spite of the mobility of our population We have followed about 90% of the children through their 4th year of age

I have already mentioned the blood samples They were collected for the virus studies not only from the mothers during pregnancy and at delivery but also blood samples from the umbilical cord of all babies and, in addition, at four months of age from any children who appear to be abnormal Full histories and data concerning socio economic, cultural and educational status of the mother are obtained, as well as the follow-up of the development of the children It is expected that the project will continue until the last children have reached the age of 12 years

## *Studies of Hearing in Neonates*

(J HARDY continues ) In addition to the standard studies that are carried out in all of the 14 institutions, each institution is allowed to carry on certain special ancillary services or studies that are related to the collaborative project In 1957 Professor and Lady Ewing visited Baltimore and Lady Ewing demonstrated her auditory screening for young children She also took one of our nurses to Manchester for training This gave us a tool

with which to follow up our children from the point of view of communication In addition we developed a modification of the Ewing test suitable for 4 months of age and for 12 months of age and tried a variety of auditory stimuli on the newborn infants

The stimulus that we found most successful for the newborn child is a clacker developed by Dr William Hardy, which gives a very clean, sharp, brief signal at approximately 60 decibels sound pressure level, with a fairly broad range of frequencies The response that we used as a criterion for 'hearing' is a reflex startle response or some modification of it We applied this test systematically to the infants at about 46 hours of age About 2000 children have been so tested thus far Approximately 98% of these children gave positive, i e normal, responses

The children who failed to give positive responses fell into two general categories First, there are the premature infants who do not respond during the first few days or perhaps for as long as two weeks but who thereafter give normal responses Secondly, there are the children who had been fed just before the test and were in that state of stupor that newborn infants sometimes assume They were extremely difficult to arouse

When we relate the "passes" and "failures" to subsequent development of communication there is no positive relation whatever Errors appeared in both directions, and actually only about 2% of the children failed to respond normally

One 'miss" is particularly significant The child had a severe secondary apnoea a few minutes after birth and required resuscitation At 4 months of age he responded fairly normally to the Ewing type of screening but at 12 months he did not respond In the nursery he had given very good auditory responses but at the age of 2 years he had not developed language A thorough going definitive audiologic study revealed a profound sensory neural type of hearing loss

In my opinion, testing of the newborn as we have been doing it is useless and we plan to discontinue it On the other hand the type of test suggested by the Ewings that involves auditory distraction and the production of noise on one side of the child or the other and the orientation response of the child, turning toward the source of sound, has proved really valuable at the 4 month and the 12 month levels

(Dr Hardy illustrated these tests by means of a motion picture The film showed two normal children one who was seriously retarded mentally, and finally a child with conductive loss with hearing levels of about 60 dB (ASA) in the left ear and about 40 dB (ASA) in the right He correctly turned to the right in response to sounds delivered on this side but not to the left He was nearly a year old at the time of the test )

We are very well aware that the development of normal communication depends not only on serviceable hearing but also on satisfactory intellectual development, on intact cerebral centers that serve the functions of understanding and memory, and on the production of speech sounds and their

use as symbols. The latter function requires both intact neural and muscular organs of articulation and also an environment which offers an opportunity to learn and stimulation to do so.

*Discussion*

DAVIS: I understand that you reject the idea of auditory testing immediately after birth but that you strongly advocate later screening at the age of a few months, perhaps six months of age.

J. HARDY: Such screening at the age of a few months is very useful. By the distraction technique of auditory screening one can tell whether a child is deviant in one of several ways. About one third of the children who failed this type of test had upper respiratory infections, either chronic over many weeks or else numerous repeated infections. Many of these children when tested with more definitive types of test proved to have significant conductive hearing losses in the first year of life.

DAVIS: The detection of these losses was certainly beneficial. They obviously had not been detected otherwise.

J. HARDY: Some of these children had their tonsils and adenoids removed and showed perfectly serviceable hearing a month or two later and did not develop later problems of communication. This accounts for about one third of the children who failed in the screening tests. Most of the other two thirds had other difficulties or abnormalities in development. Some did not have satisfactory intellectual capacity, others had motor problems arising from cerebral palsy.

DAVIS: Perhaps the hearing impairment in these children with other defects would have been detected even without the screening?

J. HARDY: The hearing impairments may not be very obvious, at least in ordinary pediatric practice. The mentally retarded child shown in the motion picture film was considered by the pediatrician to be a relatively normal child at age 1. At age 4 she revealed an IQ of about 65. This would have been recognized at school age but she would not have been identified as abnormal during early childhood.

GLORIG: May the startle response be evoked by stimuli that are not auditory?

J. HARDY: Obviously the startle response can be evoked by other stimuli. For this reason in our test situation we are quite careful to keep well away from the child, who is securely held. The clacker is operated on the other side of a screen so that there can be no draft of air from it. I believe

that in our test situation we are dealing with auditory responses Our stimulus is very substantial and children may have a variety of problems and yet respond satisfactorily to a stimulus of this magnitude

(J Hardy called on W Hardy ) Would you comment on this?

W HARDY I believe that yours is an extremely sensitive test that is appropriate for the age range between 4 and 12 months I believe it is correct to say that we have not missed a single child who has a significant hearing loss If we have they will be picked up in screening later on

We all know that a child who has an auditory problem will make quite a variety of responses initially The acoustic stimuli, human voice, that you saw used in the film were about 35 dB SPL at their lowest level The girls who produce them are carefully trained with sound level meters to hold the proper levels If there are no responses they then raise their voices Furthermore, we found by experience that even though the child was giving responses within a perfectly normal range we had to reinforce There is some learning going on here

In administering this test it is important to present the acoustic stimuli at the ear level, not above or below, or it will not work

J HARDY We found another important group of children who failed the auditory screening test at 4 months but otherwise appeared normal in all respects during the first year On later examinations however, they turned out to have severe communication problems Two of them we classify as very severe primary aphasics and a third is also aphasic but less severely so

DAVIS These aphasics must present a difficult problem in differential diagnosis I am interested to know that you found as many as 3 in a population of 2000 children As an additional complication, remember the type of hereditary deafness described by Dr Wedenberg in which the children are born with normal hearing but later the sense organ degenerates and they become deaf

HUIZING What is the lowest age at which the left right localization function is reliable?

J HARDY Dr Huizing inquires about the earliest age at which the test is satisfactory This depends on the kind of response that one is willing to accept as a criterion Some response appears while the infants are still quite young but there is a very critical period in development at about the 7th month at which the child begins to turn entirely around, a 180° turn, to locate the source of sound This makes the Ewing test very useful and simple from about 7 months to 14 or 15 months of age By careful observation one can use the test with younger infants Our 4 month-old in-

fants turn their eyes in the direction of sound and some of them give quite good head turns but not as much as 180°

We have tried using the human voice as an auditory stimulus with the newborn We obtained head turns from about 16 out of 22 newborns tested in the nursery However, testing the newborns is unsatisfactory because you have to have them at just the right level of arousal If they are too hungry and wakeful they won't turn their heads in response to sound If they are full just after feeding they are torpid and go to sleep

W HARDY By 16 weeks of age we have been able to measure or observe 15 different kinds of response to auditory stimuli It is certainly safe to assume that the auditory reflex (orientation) is well developed in the normal infant by that age

A EWING We must consider these auditory tests from two angles First there is the point of view of mass screening where it is only practical to employ workers who have had rather limited training We ourselves now train such workers to rely on what we call a decisive response This is the full rotation of the head as well as the eyes This response requires a certain degree of maturation which seems to be reached at about 7 months From the point of view of the audiological specialist we may think of workers who make audiology their profession and who have far more training and experience than the workers who conduct mass screening We would agree that such trained workers can observe and draw inferences from lesser degrees of orientation at an earlier age

CHAIRMAN This is an important point Tests that might be applicable to the population as a whole must be simple and, we hope, foolproof They must not require anything too elaborate, either in instrumentation or in the training of the observers Actually a well trained worker can make certain simple tests very effective On the other hand, someone who makes such testing a profession can make more effective and searching tests We recognize how the skill of the neurologist enters into his assessment of a patient, and the neurological examination on which it is based is not delegated to a laboratory technician

J HARDY In summary, I want to make a plea for the awareness of possible auditory problems and a high index of suspicion, and for the use of the Ewing test to verify or deny the suspicion that a child is suffering from such difficulties I also emphasize that, for mass screening, the Ewing test for children from 8 to 14 months has proved very effective and useful, particularly in relation to respiratory problems This test has been carried out in Baltimore in several Public Health Clinics by a group of volunteers who are especially trained to this, but women who are interested in hearing problems Thus at very small community expense a considerable

number of the underprivileged children who have a high incidence of upper respiratory infections have been assisted by early recognition of their problems More definitive testing is required however for the children who fail to give normal responses to sound

DERBYSHIRE An auditory test that is in use in the Mt Pleasant State Home and Training School in Michigan has interested me The observer wears ear phones and hears tones in one or the other ear The child is also given the tone in the same ear in which the observer receives it but only half of the occasions when the observer receives the tone The observer decides whether the child responds or not without knowing whether or not the child actually received a stimulus at that moment These controlled tests are very helpful but the test does require a greater number of observations

IRELAND I wish to emphasize once more what has been said about the difficulty of testing very young children after they have been fed and *want to go to sleep* Our failures with the testing of neonates have all been false negatives in newborns who were tested immediately after feeding and who apparently slept so soundly that they failed to respond

CHAIRMAN Investigators will undoubtedly continue to have good new ideas and there will be many variations in these auditory tests for young children There are many criteria that may be used beginning with the startle reflex and the eye blink and including changes of heart rate movements of the face general muscular movements and finally orientation to the source of sound I doubt whether we are ready to establish an International Standard for such auditory testing!

We shall now hear from several members of the conference about what is going on in their various countries in the way of screening infants and young children for auditory impairments and what is done for these children when they are identified In some places there are studies like Dr Hardy s section of the Collaborative Perinatal Project which are temporary experimental studies to gain new information Elsewhere there are well established continuing routines the object of which is service to the community Another of the experimental studies will be described by Mrs Downs

## *Early Primary Screening*

DOWNS Experience in observing the auditory behavior of over 5000 new born infants leads us to believe that it is feasible to screen for peripheral hearing deficits at birth (See Downs and Sterritt 1964 Identification audiometry in neonates a preliminary report *J Aud Research* 4 69 80 ) Among these we found two with congenital hearing defects and two others that seemed to present central nervous system problems I agree with

Dr. Janet Hardy that the percentage of auditory defects detected immediately at birth is certainly small and we cannot tell just how many may develop problems later

It may be possible to obtain other useful information regarding the auditory behavior of infants at birth, provided that careful quantification of observations is made If many observers are to look at these infants, a way of coding what they see must be available, so that the best interobserver reliability can be obtained The system used at the University of Colorado Medical Center is presented here for those who wish to make the same kinds of observations

Three aspects of infant testing are controlled or defined as accurately as possible 1) the stimulus, 2) the observations of responses, 3) the infant's state and condition These three aspects are treated as follows

1 *Stimulus*

Two simple acoustic signals that can be easily measured and reproduced are used The first is a broad band of white noise, which is presented at sound pressure levels of 70, 80, 90, and 100 dB, measured at 4 inches from the loudspeaker Such a stimulus regularly elicits a generalized response

The second stimulus is a narrow band of filtered noise, peaking at 3000 c/s with energy only between 2500 and 3500 c/s, presented at the same sound pressure levels 4 inches from the ear The 3000 c/s band is used to differentiate hearing losses which have better hearing in the low frequencies than in the higher frequencies It is felt that only through this kind of discriminating acoustic signal can all significant hearing losses be identified An examination of the audiograms of congenital hearing losses shows that a broad band noise would be heard and responded to by many infants with significant hearing losses, whereas a narrow band at 3000 c/s will not be heard A narrow band of noise is chosen rather than pure tone signals because complex signals lend themselves to greater accuracy of measurement and control in a sound field These acoustic signals have the obvious advantage of being measurable and replicable by any investigators who wish to communicate their findings in a meaningful way to others

(At this point Mrs Downs showed several slides illustrating the falling audiograms seen in hereditary hearing loss, in cases of perinatal pathology, and in cases of deafness following maternal rubella The point was made that whereas enough hearing remained in the low frequencies for these children to respond to a broad band of noise, the impairment for high frequencies was so great that few if any of them would respond to the filtered band of noise centered at 3000 cps )

GOLDSTEIN (in absentia) In cases of maternal rubella the audiogram is frequently U-shaped with better sensitivity at 3000 cps (Goldstein, Landau

& Kleffner, *Annals of Otology, Rhinology & Laryngology*, 1960, *69*, 756–767) In fact, this audiogram is characteristic enough to help decide whether or not a congenital deafness is hereditary

2 *Observation of responses*

(Downs continues) Rating scales and rigid definitions of response categories are applied to the various parameters of the infants' responses so that independent observers can agree on what they have seen when the very complex event of an infant's response takes place The best inter-observer reliability has been obtained through treating these parameters as separate phenomena in the following way

| Parameter | Description | Code |
|---|---|---|
| 1 Time variables | 1 Duration of response | – – seconds from beginning of response |
| | 2 Latency of response | – – seconds following signal presentation |
| 2 Site of observed response | 1 Eye | E |
| | 2 Entire body (Moro reflex) | M |
| | 3 Cessation of body activity | C |
| | 4 Limb movement | I |
| | 5 Head turn, toward or away from stimulus | T or A |
| | 6 Facial grimacing | G |
| | 7 Mouth sucking | S |
| 3 Intensity of response | 1 No response | 1 |
| | 2 Questionable | 2 |
| | 3 Clear but weak | 3 |
| | 4 Strong | 4 |
| | 5 Paroxysmal | 5 |

3 *Condition of the Infant*

The state of the infant at the time of observation is recorded as follows.

| Condition | Description | Code |
|---|---|---|
| *1 Observed state* | *Sleeping-quiet* | SQ |
| | Sleeping grimacing | SG |
| | Sleeping moving | SM |
| | Awake quiet | AQ |
| | Awake grimacing | AG |
| | Awake moving | AM |
| | Drowsy | DR |
| | Fretful | FR |
| | Crying | CR |

In addition, other background conditions which may influence the infants' responses are noted, including the exact amount of drugs given to the mother at delivery, the familial history of deafness or other problems, and the entire perinatal history of the infant

Such rating scales and numerical systems might form the basis of a Rating of Auditory Behavior at Birth Not only would such a rating identify those infants with peripheral losses, but it should also be useful in relation to conditions which are found later as the child matures

(Mrs Downs illustrated her talk by showing a motion picture film The various types and intensity of response that Mrs Downs had described were well illustrated )

DAVIS This has been a very impressive film It is my understanding that you propose to use it as a training film It should be very effective Now may I ask over what range of age these particular responses and intensity scales would be applicable?

DOWNS From birth to four days old

DAVIS I recall that in the course of screening 5000 infants by this method you detected four with auditory impairments, two of them peripheral and two of them central Do you think it is worth carrying out this kind of screening in every hospital across the nation in order to pick up such a small proportion of cases? Is the amount of equipment and the amount of training that is necessary out of proportion to the accomplishment?

DOWNS Our pediatricians have been exposed to this program now for three years One of them, in reply to just such a question, said, "We have done chemical tests on 6000 infants in order to find one case of PKU (phenolketonuria) and it is worth it" If volunteers can carry out these hearing tests it is inexpensive, it doesn't interrupt the nursery routine, and I think it is more productive of results than tests for PKU

CHAIRMAN Can someone tell us what PKU is?

W HARDY Phenolketonuria is a genetic metabolic disease If it is not recognized very early in life the child will be seriously mentally retarded If it is recognized a special diet can be prescribed which will prevent mental retardation I don't think the two situations are really comparable at all

CHAIRMAN I was going to make the same point With phenolketonuria there is great urgency for prompt detection because something definite can be done and it must be done early if it is to be done at all But in the case of hearing we might allot our time and effort somewhat differently We must ask the question whether it is important to find these children within the first six months of life Might it be done as well or better a little later? The values will have to be balanced one against the other

DOWNS *I think it is important to find them in the first six months, and* preferably by screening at birth, because this is the only time the entire population is available to us There is no other time until school age

CHAIRMAN This is one of the questions before us, whether there are better ways in which our medical care might be organized, whether formally, through state or municipal organizations, or perhaps by having a more alert medical profession to pick up these children somewhere between the delivery room and the school room

RAPIN Do I understand you correctly that the two children who did not respond to sound turned out to have central auditory deficits without *peripheral hearing losses but with language problems? Furthermore* did you find other children with brain damage who did not subsequently turn out to have language deficits but who showed abnormal responses by your tests?

DOWNS No In our screening we did find two children who did not respond adequately but who later turned out to have normal hearing but abnormalities in the central nervous system The two who proved to have defective hearing both had peripheral deficits One of them is now almost 3 years old and we have been able to confirm the diagnosis by subsequent tests The other is a son of parents with congenital deafness

WALTER Do you think it is worth introducing any sort of automation in the recording of the responses so that you could use statistical methods to relate the response to the stimulus?

DOWNS Very much so Myographic methods and other forms of instrumental recording could be used to measure the response accurately

WALTER In this case wouldn't you be able to obtain the records with a really untrained person and not have to train people to observe as carefully as at present in order to do the screening?

DOWNS Very much so, and more accurately Auditory screening of infants at birth is particularly desirable in view of the present developments of auditory measurements using evoked responses The screening tests will identify those infants with suspected problems, and the more definitive tests using evoked responses can then be applied to those children in the neonatal period It is only with such early identification and diagnosis that the question can finally be answered as to whether it will be advantageous to begin auditory habilitation at birth

GRIFFITHS We have carried out screening tests of neonates administered by volunteers on about 1400 cases in three hospitals The sound signals

were pure tone and the observers checked a list of indices for response to each signal 13% of these newborns were selected for follow-up because their response was nil or meagre When they were re-screened later over a period of months, 96 8% of the 13% were considered to have less than normal hearing by otologists Of the 1400, the percentage of those considered to have hearing losses was 0 5% This is a small percentage but we thought the effort was worthwhile

DAVIS What was your criterion in terms of hearing level? How much hearing loss did these 0 5% of the cases ultimately turn out to have?

GRIFFITHS They were not all the same. We considered it a hearing loss if the hearing level was worse than 30 dB (ASA)

DAVIS This gives us some idea of the magnitude of the problem One-half of 1% of the population turns out to have an impairment in the speech range of 30 dB or more

GRIFFITHS While the incidence of deafness or severe hearing impairment in the newborn *seems* to be low, the range of 1% to 2 0% of the current birth rate (4,000,000 in the United States) involves from 4000 to 80,000 children *per year* in the United States alone

W HARDY, SILVERMAN, DAVIS In general discussion it was agreed that some clearer definitions were necessary in order to estimate the incidence of hearing impairment among children but that the point would be deferred for the present

LING Why do you use this complex sound rather than pure tones? I know that reviews showed long ago that pure tones were not entirely satisfactory Is that why you developed complex sound as your stimulus?

DOWNS Complex sound [illegible] ind of noise that we use is [illegible] pure tones It is simpler [illegible] onset than pure tones There are many reasons [illegible] we choose it

DAVIS It does get away from the problem of standing waves The distance from a loudspeaker to the ear is not nearly so critical and noise is more effective than pure tones in at least one relevant reaction, namely the contraction of the intraaural muscles That contraction may be initiated by pure tone but it is very poorly sustained If noise is the stimulus it is much better sustained

CHAIRMAN I will now ask some of our other members to tell us what is actually being done in the way of routine screening of children in their

countries Their medical programs may be organized somewhat differently from ours and the points of view toward them may also differ

Barr In Sweden, 99% of all children are born in hospitals or special maternity institutions Up to school age the children have free health supervision at the Child Welfare Centers At these centers, 90% of the children are regularly examined during their first year of life The visiting frequency falls at higher ages, and at the age of 6 it only amounts to 20% In Sweden, compulsory school starts at 7 and from that age all children are regularly supervised by the school doctor We consequently can screen all children for deafness as newborns and during their first school year The schools have now a fully developed organization, and our interest has therefore turned to the newborn children

At Eskilstuna county hospital, Fröding has conducted an investigation on 12 000 newborn children He used a gong of 13 5 cm diameter, hanging suspended in a ring with a handle attached to it The maximum sound level was 126–133 dB relative to 0 0002 μbar with a spectrum rather like white noise 95% of the children had positive auro palpebral reflexes (movement of eyelids) Sooner or later all cases reacted with the exception of eight who were found to be deaf when followed up

At the children's clinic of Karolinska Hospital, in Stockholm, all newborn children with severe icterus, repeated attacks of cyanosis, or prematurely born children, were examined by my brother, Mats Barr, according to Wedenberg's method, the auropalpebral reflex and wakening with pure tones So far 1717 children have been examined Of these, 235 cases had either no reflex response or a raised threshold Some of the children died, and some showed a normal response after some time Eighty-eight (5 1%) still did not react normally when they left the hospital A few more of these children reacted normally during the first month of life

The investigation is still going on We definitely know that several of the children have a sensory-neural impairment, others are still under observation Especially the cases under observation are very difficult to handle and one has to be extremely careful with the parents The examination has shown a way to recognize a hearing defect early, but also shows the great need of resources for taking care of all these cases An early diagnosis without early treatment is rather meaningless

The higher standard of living, better information and consequently better observation, as well as the institution of audiology for children with 15 years of activity, have contributed to the fact that the first contact with the deaf and severely impaired children from the Stockholm area is established at increasingly lower ages *Nowadays, it is an exception if a child is more than two years old when it comes to the institution for the first time* A great number of the children have been diagnosed before the age of one year (see Table I) But there are still a number of children who come too late to their first examination, and these are mainly children

TABLE III 1 *Deaf and Hard of Hearing Children from the Stockholm Area Born 1957–1961*

| Age at time of diagnosis | Extended hearing loss (Br) >85 dB | Extended hearing loss (Br) <85 dB | High tone loss |
|---|---|---|---|
| <1 year | 15 (1) | | |
| 1 year | 15 (2) | 5 | |
| >2 years | 6 (4) | 17 | 9 |
| Total No | 36 (7) | 22 | 9 |
| Risk group | 20 | 15 | 4 |

( ) Children living in the Stockholm area, but born outside this district

who do not live in towns The anamnestic dates for these children indicate that the parents realized at an early stage that something should be done and that they even turned to various authorities This shows that information should be directed toward the medical authorities to whom the parents turn when they suspect a hearing defect All information is rather meaningless if it is not combined with the establishment of institutions of audiology that can take care of the children and parents and secure a diagnosis and an early treatment

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DAVIS What is being done in Sweden for the 5% or thereabouts of children who are identified by your screening methods as having hearing impairment?

BARR We have to follow them up, and that is very difficult to do Many of these children are severely damaged, with brain injury or malformation In the Stockholm area we can follow them up but it is very difficult outside of that area We have 100 such cases and they must be handled very carefully Most of them cannot be handled as purely hearing problems Often there is a genetic basis and a situation may be complicated by social or psychiatric problems

DAVIS Would better screening make the followup any easier?

Barr The great number of false positives is from one point of view a big problem A false positive means that the child does not react normally for sound stimuli A follow up shows that there is no hearing loss but often we do find that the child has some other defect which can explain the primary negative reaction to sound stimuli A negative response might indicate a hearing defect or a poor general neurological status The screening test is thus not only a hearing test I think it is important to work closely with the pediatricians in the follow up Actually so far they have been very interested in this test of hearing also as a routine test to observe the child s general behavior

Chairman Dr Ewing will you tell us briefly what is going on in England?

A Ewing Lady Ewing and I have prepared the following brief statement

In the United Kingdom health visitors on the staff of every city (county borough) and county health department are available after training for testing the hearing of young children Since 1951 several hundred medical officers and probably 4000 health visitors have taken special short courses in the principles methods and practice of testing the hearing of children under five Many of the courses have been held by us in health departments in different parts of the United Kingdom Some medical officers of health wish the test to be given as far as possible to all young children others only to children in risk groups Attendance at all U K health centers is voluntary The service of course is available to all parents and children As yet there are considerable variations as between place and place in the percentages of the young child population who receive the tests These relate in part to the strength of the health visitor staff Personal factors also occur affecting the desire and willingness of parents to have their children tested Publicity is given in some health departments to the advisability of every infant s hearing being tested during the first year of life The National Deaf Children s Society a parent sponsored body has contributed to this by publishing a pamphlet Your Child s Hearing (replacing If Your Child is Deaf ) both written by us at their request This is being very widely distributed Ourselves we urge the testing of all young children Statistics about causation of deafness in childhood such as those presented to the Royal Society of Medicine by our friend and collaborating otologist Mr Kenneth Harrison and by Miss Whetnall and Professor Fry in their new book show that with 30% to 40% of large groups of children in whom they have diagnosed deafness etiology and causation are unknown In this statement we are making no reference to different methodologies and ages of testing since these will be dealt with elsewhere

The largest scale regular programme with which we are familiar is that of the City of Manchester Health Department Dr C Metcalfe Brown the

City Medical Officer of Health, stated in his annual report for 1960 that he regards these hearing tests as an important aspect of preventive medicine He reported that "after special training all health visitors carry out routine tests of babies from the age of 7 to 8 months These first simple tests can be done in the homes or in the child welfare centres, and if there is any doubt as to the child's acuity further tests can be done in the special centres Should these not be satisfactory the child is referred to the Department of Audiology and Education of the Deaf in Manchester University and the child s own doctor is notified" In 1959 four health centres were used, now eleven Two health visitors are required for every session The staff attend a monthly "refresher course" Children on referral to the University Department of Audiology are tested and examined by Dr I G Taylor, in many cases more than once He ensures that all those whom he finds to have defective hearing are seen by a consultant otolaryngologist Referrals to a consultant pediatrician are made when considered necessary As regards educational management, fortnightly parent guidance has been made available up to a limit of eighty cases at any one time (It must be made clear, however, that a very considerable part of the Department's case load of more than 700 children per annum consists of referrals direct from hospital consultants, very many of them from outside the city boundaries ) Coordination, in the Department, of medical case histories with data obtained from prolonged educational management, also from reports on families submitted by our psychiatric social worker, has facilitated longitudinal studies of a fairly comprehensive nature

Collation and review of the City of Manchester pre school age referrals to the University Department during the period January 1963 to March 1964 provides the following facts and figures —

| | |
|---|---|
| Number of young children tested in eleven health centres | 3 638 |
| Total number referred to University Department with results as follows — | 97 |
| Defective hearing and educational management (after E N T examination) | 30 |
| Defective hearing and in E N T care | 28 |
| Other handicaps or defects not deaf | 6 |
| No problem | 21 |
| No appointments kept | 5 |
| Awaiting appointments | 7 |
| Total | 97 |

Thus the total number of these young children found, on examination to need either medical or educational management or both was 64 of the 3638 tested in health centres, i e 1 76% This figure of 1 76% can be compared with that for 7632 very young children reported as having been given screening tests of hearing in a previous period of two and a half years Of them, 1 4%, after referral to the University Department, were found to be suffering from auditory conditions that required either ear,

nose and throat examination and treatment with or without educational management or deficiencies of the nervous system that affected their response to sound and capacity to learn to talk

A main objective of the City of Manchester programme is of course identification of children with defective hearing or other handicaps affecting linguistic development at the earliest possible age ideally during the first 12 months of life How far this was achieved in the 1963-64 cases under review is indicated by the following breakdown of the 64 children into groups —

| Age at first screening test | Number of children |
|---|---|
| Under 12 months | 1 |
| One to two years | 12 |
| Two to three years | 14 |
| Three to four years | 10 |
| Four to five years | 11 |
| Total | 64 |

It is clear that although the present Manchester system does not involve testing all live births or all children in any one age group before admission to school it does result in identification of a significant number of very young children who present impaired response to sound At an earlier stage in use of our screening tests from 1954 to December 1957 in the City of Leicester Public Health Department 5494 children were tested and 13 were ultimately found to have defective hearing after a second and third screening test followed by examination in the City Audiology Clinic and by an otologist Six of these last children were stated to have merely an island of hearing

CHAIRMAN Thank you for a very interesting and helpful presentation Do I understand you to say that you believe you are identifying practically all of the children with hearing impairment by the age of 5 or 6?

A EWING It is difficult to give a precise answer because the onset of deafness occurs at different ages among children before their admission to school Actually the figures we have for pure tone screening tests administered to children on admission to school or soon after it show a higher incidence than we are as yet finding in our earlier mass screening Our earlier screening we regard as a beginning but an effective beginning of this kind of thing

CHAIRMAN Dr Røjskjaer will you tell us how the situation is handled in Denmark?

RØJSKJAER Denmark is a small country with a quite homogeneous population We do not screen for hearing defects in infancy in Denmark

but despite that we believe that our audiological organization, which includes the whole country, brings almost all cases to our knowledge in a very early stage of the disease, that is before the age of two, and therefore immediate treatment can be started if indicated

Our audiological services, including the medical aspects taken care of by full-time audiologically trained otologists (oto audiologists), are organized on a state-wide basis

Although we must consider a programme consisting of screening procedures to be used in risk groups of infants, our health visitors who see all newborn infants at regular intervals could be used (Ref C Røjskjaer and Sv Bognsen Danish Audiology and its institutions *International Audiology*, 1964, *3*, 5 )

CHAIRMAN You have the advantages of a homogeneous situation and a philosophy of state responsibility for health and welfare Some people on the outside call this "socialized medicine" and the "welfare state' Is this correct?

RØJSKJAER Well, everything in connection with audiological examination and treatment of children and adults is paid 100% by the state

CHAIRMAN Well, that is certainly a different situation from what we find in some of the other countries Dr McHugh, how are these things done in Canada?

MCHUGH I speak particularly for Montreal and Eastern Canada We have not yet established any national program by any means There is no testing of the newborn in Montreal hospitals on any organized basis, but we do have at Montreal Childrens Hospital a group of pediatricians who have become interested in disorders of communication and they are testing all of their children on their own initiative This is not yet an organized program, however

Within the school system I have initiated a testing and screening program I refer specifically to the English-speaking members of the Protestant school board There are some 65,000 students in the system and for the past seven years all of those in the first and second year have been screened All of those who failed two successive tests, and as a matter of fact as far as possible those who have simply failed their first test, are referred to their family physician for examination

We tend to catch most of those who have impaired hearing We have been interested and active in the problems of hearing impairment in children for the last 15 years and doctors in the area are now quite well aware of it I believe that they actually do refer to us most cases by the age of 2 or 2 1/2 years in which they suspect that there is a lack of hearing because the children are not developing speech normally

CHAIRMAN Dr Huizing and Dr Tervoort, what can you tell us about the situation in Holland?

HUIZING I am not able to give any statistics for our country

TERVOORT I cannot give any figures for the whole country either and I can t even give the figures for my own institution, but I do feel that we are sailing somewhere between socialized medicine and the completely free enterprise system I think that most of the pediatricians and public health authorities in the Netherlands are now well aware that the very early discovery, screening and treatment including educational treatment of a deaf infant is very important We get at most of the cases long before the school age, thanks to the efforts of many interested individuals

As far as our school is concerned, we see children suspected of hearing impairment for about a whole week from Monday to Friday During that observation time many specialists come and see these children We have had many as young as 7, 8 or 9 months of age

CHAIRMAN Dr Whetnall, can you tell us how things are done in your city (London) and how the early screening might be improved?

WHETNALL I am an otologist I am not primarily concerned with training but with the results of training

It is my impression that far too many children are not found early enough A great effort was made by the Ministry of Health, and as a result the number of infants who were identified as suspected of "deafness" increased from 34 to 87 over a corresponding period of time This increase was the result of information that was sent around to all the hospitals and clinics dealing with deafness, advising people what to do, where to go, how to deal with the problem

The main difficulties are first, a lack of money and second, a lack of a sufficient number of trained workers, particularly a lack of people to do the tests in the first year of life This screening and informing is still not routine in all counties in Great Britain Some *are* doing it and the number is increasing but there must be a tremendous propaganda and more information directed to the general public and also to the people who are doing the screening What is needed more than anything is to make people aware of the importance of the infant's hearing and to determine whether it is developing as it should *during the first year of life*

CHAIRMAN These are all important points, and they lead us directly to the question of just what we should tell the people whom we wish to alert, whether they are the physicians or the parents

WHETNALL A word also about the tests I am responsible for making a diagnosis The children who are brought to me are suspected of some impairment The parents are already very concerned and I cannot be too

vague in my statements to them. I never attempt to make a diagnosis on a single visit but I must make it sooner or later

I have used chiefly Ewing's test but I have modified it by examining normally hearing children to see how the responses change with age I consider *it essential to continue to determine whether the reflex responses* are still present, not merely to look for them during a particular month of age Turning toward the source of a quiet sound is not a very brisk response at the age of 4 months I don't think there is an exact month at which it appears but it is usually well established by 7 to 9 months

Following the fourth month, before clear localization develops, the infant will turn and look at a new sound but it will only look once The child will turn and look toward the sound but will not look *at* it Because the child will only look once it is essential to test opposite ears, that is, you start each new sound with the ear opposite to the one with which you started with the previous sound (Dr Whetnall illustrated with several slides )

In a group of 87 babies referred for testing in 1962 I found that 24 *were* deaf and 63 were *not* deaf The wrongly suspected infants had, without exception, been tested or suspected at the age of 3 to 4 months At this age the infant may normally show no reflex response, and there may not yet be any response to quiet sounds A number of infants at risk who were *tested at this age were thought to be deaf*

The development of responses was delayed in babies who were premature and also followed, in a number of cases, where there was some illness or operation The mentally retarded children were either dull, severely retarded or ineducable and three of them had motor disability in addition (Dr Whetnall illustrated the responses of infants and also their audiograms with a series of slides ) If you find that a child at 11 months is only looking once at a sound and not again and is also unable to sit up it is almost certain that the child is retarded and that the retardation is preventing the learning process

CHAIRMAN (to readers of this volume)

For clarity and in spite of some repetition we insert here the text of a statement submitted by Dr Whetnall after the conference

## *Developmental Tests of Hearing*

WHETNALL Tests of hearing depend on a knowledge of responses to sound in the normally hearing infant especially during the first year of life These tests depend on the type of response given rather than the distance at which a sound is heard, this indicates the stage of development in hearing that the infant has reached Hearing has a peripheral and central mechanism For this reason pure tones alone are inadequate in estimating an infant's or young child's hearing as the responses to pure tones give no information about the ability of the infant to use the central mechanism




FIG III 1 *a* 3 months startle response
FIG III 1 *b* 3 months no response to quiet sounds

At birth the response to sound is reflex (Fig 1) By the age of 3 months this reflex starts to become inhibited as the central mechanism takes over control of auditory responses and by 8 to 9 months it can no longer be elicited The generalized muscular jump disappears at about 4–5 months, but the eye blink takes longer to disappear completely

While reflex responses are becoming inhibited, the infant at the age of about 4 months will start to respond to the quiet sounds which he hears about him The infant characteristically will look once towards a sound



FIG III 2

which is new or which is not familiar there is no response if the sound is repeated (Fig 2) So it is advisable to alternate the ear first tested with each new test sound Localization occurs about the age of 8 to 9 months as the result of learning (Fig 3) The infant then turns and looks at a sound which is understood and is familiar Speech is the most important sound




Fig III 2

of all and the infant will not only turn and look but will also try to imitate what he hears

By one year understanding of simple sentences is beginning during the following year single words are used and later usually by 2 years put together to form sentences



Fig III 2

which is new or which is not familiar there is no response if the sound is repeated (Fig 2) So it is advisable to alternate the ear first tested with each new test sound Localization occurs about the age of 8 to 9 months as the result of learning (Fig 3) The infant then turns and looks at a sound which is understood and is familiar Speech is the most important sound

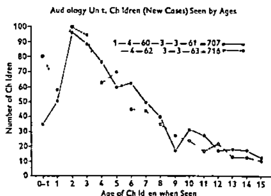



FIG III 4

The diagnosis is made by a delay or absence of the responses which are found in the normally hearing infant Both reflex and comprehension hearing are affected Sounds which should cause the infant to look up and around will produce no response unless close to the meatus The response to sounds typical of the infant aged 4 to 5 months may still persist at 8 months or more

Congenital deafness is central as well as peripheral and the stage in which sounds are heard but not understood persists beyond the normal age, while lack of understanding persists, speech will not develop Failure to recognize this may result in a failure to give the young child the right conditions to learn, which are that the sounds of speech must be heard (A) loud enough—(2 aids), (B) often enough, and (C) at the right age, at the time the normal infant learns

These tests are of value in making a differential diagnosis of deafness This graph shows the increase in the number of infants under 1 year who were seen following propaganda by the Ministries of Health and Education in Great Britain (Fig 4) Not all of these children, however, were deaf (Fig 5)

The reasons were as follows

*Not Deaf Infants under One Year*

| | | |
|---|---|---|
| 1 Wrongly suspected | | |
| Lack of knowledge of tests of hearing | 32 | (all aged 3 to 4 months) |
| 2 Inhibition of response | 3 | |
| Separation | | |
| Lack of individual attention | | |
| 3 Delay in learning to hear | 6 | |
| Prematurity | | |
| Delayed maturation | | |
| Severe illness | | |
| 4 Retarded | 22 | |
| Specific (locomotor and hearing) | | |
| Backward very backward or ineducable | | |
| | 63 | |

*Nuffield Hearing and Speech Centre — London Infants under One Year*

| | 1962 | 1963 |
|---|---|---|
| Deaf | 24 | 48 |
| Not deaf | 63 | 46 |
| Total | 87 | 94 |

Fig III 5

It will be seen that the diagnosis of aphasia is not applied in any of these cases It is very rare, none in this group It is considered to be a diagnosis without meaning, except in a few cases as the result of trauma where a child has already learned to talk and lost his hearing

CHAIRMAN Now our discussion has really moved across the borderline from the first-order screening done to identify the young deaf child, to the second-order screening in which we try to make a judgment such as the otologist must make

A EWING In regard to localization, a child psychologist, Dr Ruth Griffith in England, has reported tests of 571 children under 2 years of age She describes what she calls the "abilities" of the babies She reports that the norm is that at 7 months of age a child should look or turn his head when the examiner calls softly, "hello, hello"

My wife, Dr Taylor, and I are constantly testing normal children because we have to train large numbers of medical officers and health visitors to carry out these screening tests We are impressed by the great variation in the rate of development among children of these ages, although as yet we do not have accurate data to show the distribution

We do see children who can respond in this way at a much earlier age and we also see children who do not do it, even rather later, although they seem to be quite free of any auditory disorder or impairment

## *Maturation*

CHAIRMAN Dr Hawke, do you have any comments from the point of view of psychiatry ?

HAWKE I am impressed by the statement of Dr Ewing regarding maturation Certainly in the early stage of neonatal development you are dealing with pretty primitive responses at, so to speak, a low phylogenetic level A series of studies, particularly of the motor function, has shown that the neonate has certain patterns such as the Moro reflex which vanish with maturation As the higher centers mature, the primitive functions gradually disappear and are replaced by more complicated ones

We have been particularly interested in the broad problem of maturation and we feel that there is a tremendous variation in the development of

specific skills in individuals We know this in reading and spelling, and many of you see it also with the children who come to you as so-called "congenital aphasics" who are not the least bit deaf but have inadequate perception

It is our feeling that there must be a significant number of children who have a delay in the maturation of auditory reception, a group of children who are often first considered to be deaf because they show little response to sound and then are considered to be aphasic because they do respond to sound but they don't respond to the voice Ultimately they develop beyond what you would expect with an aphasic, and it seems probable that they represent a significant maturation lag in auditory perception The specific defects are not correlated with general intelligence Their distribution in the population is entirely different from that associated with intelligence

SILVERMAN Are you saying, Dr Hawke, that we should be very cautious about predicting because of these different rates of maturation and that our thinking should not be too deterministic?

HAWKE I am quite certain of that In my practice I see many children with reading disabilities and children who are sent to me as aphasic with "non-language" At one time we talked about a lag and said that this was a delay in maturation and that eventually the child would develop normal speech But over the years I have found that my predictions are inaccurate and that these lags often persist Some of the children eventually do talk although they didn't talk at 3 or 4 years of age, but as adults or as older children they have tremendous difficulty in phrasing Their speech is awkward, almost aphasic If you have a precise test I think you should be able to pick them up but they are often submerged in the general population With all of this in mind one has to be very careful in making predictions

J HARDY I agree with Dr Hawke, and I would add that the delay in apparent maturation can sometimes be environmental in origin Let me cite a case to substantiate this, a baby of about 5 months of age that we screened quite early in our series He gave no responses to sound and we referred him to Drs William and Miriam Hardy for a more definite work-up, using the electrodermal response and other types of more definite test On these he did respond but in a peculiar fashion, and we came to the conclusion that this child suffered from an early central communication problem We wanted to see him again at 8 months for further testing The home visitor went and asked for the child, who lived in a crowded home, and was told by a person living at the address that he didn't know such a child existed However, the mother was across the street in the corner bar They found the mother and she took the home visitor back to the house and showed her the baby who was in a third floor room

in a high cardboard box where he spent all his time He was obviously poorly cared for and not stimulated at all Shortly after this the case was reported to the proper authorities as neglect and the baby was adopted by a woman who was very interested in children By the time he was a year old he was learning language and his electrodermal and other tests were perfectly normal This is one child who had no opportunity to learn

We also had another child recently who came to us with no language at the age of two He had been cared for by a 5-year-old sibling most of the time This is another child who seems to be normal but who has had no auditory stimulation

WALTER I want to reinforce what Dr Ewing, Dr Hawke, and Dr Hardy have said about the lack of information concerning the maturation of the nervous system, and with particular reference to the cranial reflexes A good deal is known about the spinal reflexes and we have general milestones of development, but there is really nothing of statistical value known about the maturation of cranial reflex behavior in man This is partly due to the enormous variability of the diffuse projection system in the brain It is upon these that we depend for many of the reactions we have described today I don't think there is any information at all about the histology or the functional anatomy of the maturation of that system Until this is worked up, by prolonged and arduous study, I think we shall still be working in the dark and will have to depend upon qualitative and rather intuitive statements and judgments

### *Copenhagen Conference on Neonates*

CHAIRMAN In closing our discussion of testing the hearing of neonates for purposes of identification, I will ask Dr Røjskjaer of Denmark to tell us about a recent conference dealing with exactly this same topic held in Copenhagen, immediately after the VII International Congress of Audiology in August of this year Dr Røjskjaer was the chairman of the conference

RØJSKJAER The "Neonate Conference" was organized by the Danish Hearing Centers They invited a group of some 25 professional people, a number of whom are present at this conference today, and representing various interests and points of view, to discuss the problems of neonate hearing The conference lasted for three hours and was held on the afternoon immediately following the VII International Congress of Audiology in Copenhagen In addition to the invited group more than 100 interested listeners were present in the conference hall

Three sessions of an hour each were devoted first to the testing of hearing of the foetus, second to tests applicable to the newborn infant, and third to various hearing problems revealed by such tests

Because of the considerable overlap both in the persons attending and

in the similarity of topics to our conference today, it is not necessary to review in detail each of the presentations I shall stress only those that present information or points of view not fully represented in our own proceedings today

The discussion was open by Dr H Davis (U S A ) who, as he did today, asked why it is important to examine the hearing of newborn children Very early testing may be justified if it is important to institute treatment or special management immediately Otherwise perhaps the testing can wait for a few months or even longer, when it can be done more reliably or possibly more economically On the other hand, some recent biological experiments indicate that complete sensory deprivation during the first months of life may interfere with the normal development and organization of sensory systems If this theory is correct, then an early diagnosis may be very important

Dr Wedenberg and Dr Johansson (Sweden) described the detection of the response of the human foetus to tones delivered through the mother's abdomen Dr Wedenberg has just reviewed this material for us today, so that I need say no more on this topic Dr K P Murphy (United Kingdom) pointed out, however, that great care must be taken in interpreting such tests that the mother does not hear and react to the auditory stimulus in some way and thus indirectly, through changes in her own blood pressure, heart rate, etc , induce some response in the foetus that might be interpreted as an indication of foetal hearing He also noted that the positive responses assess only cochlear function and they do not exclude the possibility of lesions high in the brain-stem

In general discussion it was pointed out that histological studies of the cochleas and brains of children with congenital hearing diseases will be of great value Also the question was raised, as it was today, but not completely answered as to the possibility of causing a traumatic lesion in the cochlea of the foetus by the auditory stimuli employed

In the second session, dealing with the testing of hearing in neonates and infants, Dr G Beckmann (Germany) showed a film which demonstrated clear reactions of hearing in an ancephalic infant The point that he stressed was that reflex auditory responses in the newborn do not guarantee sufficient central function for the development of speech

Dr Mats Barr (Sweden) and T Söhoel (Norway) discussed the results of neonatal tests of hearing with particular emphasis on the auro palpebral reflex described for us by Dr Erik Wedenberg Both of them stressed the value of screening newborn infants of the high risk group About 50% of the children with hearing defects were found in this group Only 0 1% of the children of the general population failed to pass the Wedenberg test The speakers considered these tests to be reliable and satisfactory screening tests, suitable for the neonate period

Dr Griffiths (U S A ) told us how she tests the hearing of newborn infants with an instrument called the Hearometer A small loudspeaker is

put in position over the sleeping infant and interrupted pure-tones at various frequencies and intensities are delivered from it Among other things the endpoint, that is, a satisfactory response, is the awakening of the infant In many ways the test resembles rather closely the one described for us today by Dr. Wedenberg The number of infants who do not respond positively is small, less than 1%, but those in whom hearing appears deficient are immediately given the benefit of amplified sound in order to promote the normal development of hearing as far as possible

Dr Griffiths, Dr Whetnall, Dr. Bordley and others described the various responses of babies, both asleep and awake, to auditory stimuli. The descriptions and much of the discussion were very similar to what we have heard this morning Dr Bordley (U S A ) based his presentation on the same material that was discussed for us here by Dr. Janet Hardy Dr Whetnall (United Kingdom), in the final session, made the point that the main reason for estimating the ability of infants to hear is to make a correct prognosis about the development of speech The degree of hearing loss, she said, is not the most important factor however great it may be, provided it is not total The greater the intelligence of the child, the less hearing is necessary and the ability of the mother to provide the essential conditions for the learning of speech is a vital factor

Dr Fabritius (Norway) mentioned a new birth registration form which is about to be introduced in Norway With its help it should be possible to pick up very readily the children at risk for hearing impairment and to test them soon after birth

Dr G A McCandless (U S A ), now associated with Mrs Downs in Denver, has described a modification of EEG audiometry which gives great promise for early detection of impairment of hearing A computer system is used to extract small evoked potentials from the normal on-going activity of the brain I hope that we shall hear more about this technique and its possibilities today Dr. McCandless reported that with this technique it would appear feasible to perform certain tests on children and on adults and that he could obtain clear auditory responses at levels within 10 to 20 dB of normal hearing levels. Dr Davis (U S A ) stated in discussion that he was having great success with the same method applied to older deaf children but that he had not extended his studies to infancy

CHAIRMAN Thank you for a very concise and informative report on the Copenhagen Conference on neonate hearing Are there any of those present who would like to add a comment?

DOWNS I would like to call attention to Dr Rita Eisenberg's paper, presented to the VII International Audiological Congress. She reported on a study of neonatal responses to various sounds and noises It has been published in the most recent issue of the *Journal of Speech and Hearing*

*Research* and I recommend it to all of you as a very fine study of hearing in the young child

*Reference*

EISENBERG R B., GRIFFIN, E J., COURSIN, D B., and HUNTER M A., 1964 Auditory behavior in the human neonate, a preliminary report *J Speech Hearing Research*, 7, 245–269

## B. THE ETIOLOGY OF NEONATAL DEAFNESS AND THE "HIGH-RISK REGISTER"

CHAIRMAN During the discussion of the value of neonatal testing screening for impairments of hearing, Dr Janet Hardy introduced the concept of a "High-Risk Register" which would be the basis of positive action that can and should be taken during the perinatal period The establishment of such a register would largely offset the argument made in favor of perinatal testing that the delivery room and nursery offer the only opportunity to screen the entire population of young children Dr Hardy pointed out that most of the cases of impaired hearing are found in particular groups of children who can be identified in advance on the basis of family background, the mother's pregnancy, conditions of delivery, and events of the immediate post-natal period Dr Hardy proposed that all of the children falling in these groups should be listed and obstetricians and pediatricians should be alerted to follow up these children with particular care It was Dr Hardy's contention that if all of the children listed in such a High-Risk Register were examined periodically with the risk of hearing impairment in mind, very few cases would actually be missed

The concept of a High-Risk Register was received with enthusiasm by the conference Dr Hardy, during the lunch hour, drafted a list of recognized causes of neonatal deafness and wrote it on the blackboard The various items on the list were discussed at some length during the early afternoon session and several items were added to Dr Hardy's original list

The High-Risk Register in its final form, together with a brief commentary drafted subsequently by Dr Janet Hardy, will be found in the summary, on page 15 It constitutes a list of what we recognize as known causes of deafness in young children

The discussion of the High-Risk Register was animated but rather difficult to reconstruct from the tape recordings Some of the more definite questions and comments are the following.

WEDENBERG The most important cause of prenatal exogenous hearing impairment is maternal rubella during the first three months of pregnancy. Preventive measures are here an important matter It is open to question

whether girls should not be exposed to the rubella virus at non-fertile age If there is a risk that a pregnant mother might be infected in the first three months of pregnancy she should be given gamma globulin or, preferably, immunization serum, to give this after the symptoms have appeared is futile The toxicity of the rubella virus varies from one epidemic to another It was shown by Barr and Lundstrom (1958) in a Swedish series that 4% to 7% of the children suffered from severe hearing impairment—that is, a hearing loss of more than 55 dB for the better ear, thus the risk was one hundred times greater than the figure of 0 7% for the normal incidence of deafness in Swedish children Since 1963 in Sweden the occurrence of maternal rubella during the first 3 months of pregnancy has constituted grounds for an abortion

The commonest causes of perinatal hearing impairment are (1) prematurity, (2) asphyxia, (3) birth damage, and (4) icterus neonatorum (RH- or ABO incompatibility)

The prevention of prematurity presents a difficult problem, and obstetricians are working hard to solve it

Asphyxia can occur in various ways One important cause may be the administration of 100% nitrous oxide during labor The proportion of oxygen to nitrous oxide should be 60 40, or, for long periods, 80 20 Asphyxia prior to delivery may be promoted by the uterine contractions Radioisotope measurements have shown that during labor with normal contractions the circulation in the placenta decreases to 1/5 of the normal (Caldeyro Barcia, 1958) Short delivery is characterized by contractions of great intensity, long delivery by ineffective labor and, finally, long contractions, in both cases there is a tendency to expose the child to oxygen deficiency For a firstborn a normal delivery should not take less than 4 hours or more than 24, and for a multipara it should not exceed 15 hours Today, an obstetrician is able to guide the delivery by manipulation and with drugs

A severe form of asphyxia immediately after birth is asphyxia pallida neonatorum Such children formerly died Westin, Nyberg, Miller and I (1962) have treated these children by hypothermia If the respiration has not started within 5 minutes of birth the child is placed in water at 8–10°C, whereupon the body temperature falls to 23–27°C and the oxygen needs are reduced The respiration then gradually begins The longest that a child has lain in cold water without the respiration starting is 52 minutes In severe cases of asphyxia with cardiac arrest, hypothermy therapy has been supplemented by transfusion of oxygenated blood

In all the infants, auditory tests given after recovery indicated normal hearing This was an important finding since at birth the hearing is the most sensitive of the senses to oxygen deprivation The fact that the hearing was normal at the first measurement suggests that thanks to the hypothermia the supply of oxygen had been sufficient and that the child was normal also in other respects The first children to receive this treat-

ment are now 5 years old and completely normal The first hearing test (APR and awakening) was of great prognostic value in these cases

When the bilirubin in neotal hemolytic disease is 20 mg percent a blood transfusion is given Even in cases in which yellowing has shown that the process is far advanced the child has recovered after repeated transfusions without signs of hearing damage.

## *References*

BARR, B., and LUNDSTROM, R., 1958 Deafness following maternal rubella *Acta oto laryng*, 53 413

CALDEYRO BARCIA, R., and POSEIRO JUAN J., 1958 Fetal and maternal dangers due to misuse of oxytocin *Congres International de Gynecologie et d Obstetrique, Montreal, 1958 Tome II*, 450

WEDENBERG E., 1956 Auditory tests on new born infants *Acta oto laryng*, 46 776

WESTIN, B., NYBERG, R., MILLER, JAMES A., JR., and WEDENBERG E., 1962 Hypothermia and transfusion with oxygenated blood in the treatment of asphyxia neonatorum *Acta Pediatrica Suppl* 139

## *Congenital deafness*

BERTRAND I would like to mention a type of congenital deafness that apparently depends on an inborn error of metabolism As an otologist in our Speech and Hearing Center I see a considerable number of deaf children and I must admit that we have to classify something like 40% of them as being of "unknown origin" About two years ago, we noticed that a certain pattern of deafness seemed to appear frequently in three particular families The children in these families appeared to have normal hearing at birth and it is only toward the 18th month of age when the deafness starts to occur We looked up the literature but found that deafness apparently had not been reported in relation to errors in metabolism Nevertheless we undertook a series of biochemical examinations in these three families

We investigated one of these families very thoroughly from this point of view There are seven children in the family, six of them are deaf The six children presented the same pattern of deafness, which began to appear at the age of 1 1/2 to 2 years The seventh child is not deaf but, following a very careful investigation by our social worker, we believe that there is some question as to the paternity of this seventh child We measured amino acids and certain enzymes, and the results are very distinctive in the six deaf children of this family and seem to point clearly to an inborn error of metabolism

In one of the two other families there appears to be a sex-linked deafness which is transmitted to the boys There are two boys who are deaf and a girl who is not deaf The mother is not deaf but she presents a biochemical abnormality We do not know just what the relation of the deafness to this abnormality may be, but we have a biochemist working on this project and we are now doing routine tests on blood samples and urine samples of all of the children who are coming to the Speech and Hearing Center.

As an example of a specific inborn error of metabolism which is proven, accepted, and curable, I can mention phenolketonuria This occurs in something like one case in 5000 or 6000 Phenolketonuria is not a basis for deafness but it is an example of an error of metabolism which adversely affects the nervous system The error of metabolism that we found is not new but it has not previously been brought into the broad relationship that we are looking for in relation to deafness

CHAIRMAN This represents one form of real progress One more variety of deafness, one more specific cause has been identified just as we have already separated out rubella and the Rh incompatibility In this way by degrees we shall reduce the percentage of cases of unknown origin These congenital and hereditary factors are very important from the point of view of the high-risk register

*Complications of labor*

J HARDY Complications of labor include both prolonged and precipitate labor and, most important of all, premature delivery 75% of the premature babies in our collaborative study behaved abnormally in some way, and it is my impression that a large number of babies weighing less than 2000 grams at birth have problems in communication I'm not sure why this should be, but hyperbilirubinemia is very common in these babies under 2000 grams and this may be the basis of the difficulty Apnoea is common in these babies also and this may be another reason In any case, prematurity is an important item in the register

One way in which difficult labor may cause difficulty is by occlusion of the basilar artery, perhaps because of undue traction on the head by forceps The medial geniculate body and other centers which are important pathways in the conduction of auditory impulses are among the first structures to suffer in this kind of injury

*Blood incompatibility, particularly the Rh factor*

RAPIN Do you mean blood incompatibility, apart from neonatal jaundice?

J HARDY I think it is helpful for obstetricians and pediatricians to think about it both ways During the prenatal period we don't know what the bilirubin level is going to be

RAPIN In our own review of children who actually have language deficits and hearing losses we found in some charts the diagnosis of blood incompatibility In going back to the hospital records it turned out that a few of these children, although they had a different Rh group from their mothers, were not jaundiced and were not transfused I therefore wonder whether there may be some risk from the blood incompatibility

J Hardy I suspect that blood incompatibility without jaundice is not a risk, but I am not sure

Rapin This is my impression also

J Hardy. In the High-Risk Register I have indicated a level of 20 milligrams per 100 cc of bilirubin as constituting a high risk We should probably think of the risk beginning at lower levels of perhaps 12 milligrams per 100 cc, although approximately 10% of our population actually have bilirubins up to this level

Barr I would like to stress the importance of the bilirubin There are several kinds of bilirubin and some are dangerous and some are not dangerous We should remember also that a premature baby that is blue and anemic may also have a very high bilirubin level but this is overlooked because you can't see the jaundice

J Hardy There are certain drugs in wide pediatric use which increase the bilirubin level, which is, as you suggest, the more dangerous situation It is important to avoid using these drugs, particularly in premature children but I think also in all newborn babies

Barr Yes, the sulfas for example

J Hardy The sulfas particularly, and also the vitamin K drugs except for some of the newer synthetic ones

Hawke We have run through a series of children who showed bilirubin levels of 20 milligrams and found, up to 5 or 6 years of age very, very few changes Nevertheless I am really not surprised at the 12 milligram level because I believe we may have multiple effects The bilirubin affects metabolism notably phosphorylization If there is also anoxia associated with the hyperbilirubinaemia, or hyperglycemia, or an elevated temperature which places higher metabolic demands on the cells—any or all of these conditions may potentiate the bilirubin It is because of these many factors that some children with high bilirubin show no changes and others with lower bilirubin levels do show impairments of hearing

Tervoort When you speak of blood incompatibility does this refer to different races of the parents? I ask this question because since September 1st I have seen 12 deaf blind children and I was amazed to discover that 5 of the 12 were born from mixed racial parentage either the father or the mother was Dutch, the other was Indonesian

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We borrowed a sound pressure meter from Dr William Hardy and got his advice as to how to use it We found that in this variety of incubator, made from 1960 until quite recently, the sound pressure level at the place where the baby's ear would be located was 83 dB for one volume of air flow and 93 dB for another This was a noise of wide acoustic spectrum

GLORIG Was this the overall level on the C scale of the ordinary sound level meter?

J HARDY Yes

GLORIG You would get a lot more information if you measured with the A scale

DAVIS It would be still better to get a new sort of incubator

J HARDY We *did* get a new incubator We never used that particular variety again because we knew that men in industry may develop hearing losses when exposed consistently to levels of 85 dB for 8 hours a day, and here the babies were being exposed for 24 hours a day The manufacturers, of course, were extremely cooperative and redesigned the incubator and have eliminated the risk as far as their incubators are concerned

DAVIS This noise risk sometimes turns up unexpectedly We have had to do something about the air conditioner in our animal farm because the chinchillas are showing some signs of changes in their hearing thresholds

GLORIG There is a great need for an educational drive to alert to the possibility of communication disorders in young children those groups that are particularly in a position to encounter such problems I think particularly of state, county, and local medical societies, especially the sections on ENT, pediatrics, obstetrics and general practice, governmental agencies including state, county, and local health departments, welfare agencies such as family service, visiting nurses, and social workers, staffs of well baby clinics and of day-care nurseries for underprivileged children, and finally, even parent-teacher associations

I believe we should circulate the report of today's discussions to medical societies, health departments, special education departments, and audiologists

Films such as we have seen today should be made available to the general public, and to medical and para medical groups And of course there should be more articles in the popular literature, for instance in the Reader's Digest or the Saturday Evening Post

A questionnaire is needed for obstetricians, pediatricians, and residents in both fields, as well as for parents, that should include a check list of

items of behavior with rules for scoring to indicate the need for follow-up of the suspects

Finally, we should circulate very widely a description of simple screening procedures which may be used to tag an infant as a possible deaf child, and to categorize those who require specific follow-up procedures

CHAIRMAN This concludes our discussion of the High-Risk Register This register provides a list of the etiologies of hearing impairment in young children, with the notable exception of the category "Etiology Unknown" which accounts for some 40% of the children who ultimately display hearing impairments On the other hand, the High-Risk Register constitutes a practical recommendation of how to deal with the problem from the point of view of Public Health It represents a compromise to be sure, but if we can actually succeed in establishing such registers and alerting obstetricians, pediatricians and parents to the possibility of hearing impairment in this group of children and the desirability of detecting it in order to institute proper and helpful management, we shall have made a very significant contribution to the solution of our overall problem The step will now be to persuade the appropriate authorities to institute such High-Risk Registers, or, in situations where no such ready-made "authority" exists, to institute such registers by voluntary organizations Perhaps a High-Risk Register can be operated by willing volunteers just as much of our present neonatal testing is carried out by dedicated non-professional personnel

(These remarks by the Chairman are one of his "afterthoughts")

## C. THE YOUNG CHILD DIFFERENTIAL DIAGNOSIS

### *Differential Diagnosis*

CHAIRMAN Let us now pass from the neonatal period and the High-Risk Register to the problems that confront the pediatrician or the otologist in his office when a child is brought to him suspected of deafness by the parents because he has not developed speech normally, or perhaps a child whose behavior and development are clearly abnormal and in whom deafness may be one of the complicating factors The problem now becomes one of differential diagnosis Let us consider, on the one hand, what the conditions are that may be confused with deafness or perhaps combined with it and how the clinician, by means of careful observation and relatively simple tests may perform the next step, whether we call it identification of the deaf child or differential diagnosis

Dr McHugh I believe you have a motion picture film which illustrates some of these problems

McHugh The film which I will show in a few moments was not prepared for such a sophisticated audience as we have today but it does attempt to show a method of evaluating the hearing of young school children Actually the average age of the children who have been referred to us for such testing and a possible diagnosis is about 2 5 years The usual reason why they are brought to us is the lack of normal development of speech and verbal communication The average age of 2 5 years is the same that Dr Whetnall has mentioned

I think that we will all agree that by the time a deaf child is 2 5 years old he has developed a pattern of behavior that is easily recognized by anyone who has had experience in this kind of observation The method of observation that I shall illustrate emphasizes the very inexpensive equipment in the form of calibrated noisemakers that may be used for this purpose We can determine the intensity and the frequency of the noise and from the responses of the children to them we can obtain an approximate audiogram These simple methods are appropriate for pediatricians and otolaryngologists and general practitioners and even for clinics where soundproof rooms and elaborate equipment are not available

(The following is the sound track of the film shown by Dr McHugh )

## *The Problems of Differential Diagnosis in Children with Communication Disorders*

In the investigation of children who have failed to develop normal communication skills it is vitally important to recognize that an infant or pre school child may not respond to auditory stimuli for reasons other than deafness Early diagnosis is important but an unqualified hastily applied label can become an unquestionable deterrent to the child s total potential development

The first step in the early identification of children with hearing and/or communication disorders is a careful medical history and complete physical examination by a physician or team of medical specialists whose competence includes a basic understanding of these particular children and their specific developmental problems Continued medical supervision of these children must be part of their management and rehabilitation programmes This is particularly so if the child is placed on the High Risk Register

The early diagnosis of these children may be extremely difficult Multiple handicaps are common and often a programme for management must include continuing diagnostic evaluations A tentative clinical diagnosis can often be made on the basis of the distinctive behavioral patterns that are recognized for each of the four major groups of children included in these categories (*a*) the deaf (*b*) the brain injured (including those with central nervous system dysfunction aphasia etc ) (*c*) the mentally retarded and (*d*) the autistic and/or psychotic

The behavior of the congenitally deaf child is so typical that it may be used as a frame of reference in the differential diagnosis of these other children The study of deafness, or impaired hearing, in children must take into account possible defects in the central auditory system, in addition to impairment of the peripheral sensory input Damage to this complex auditory system in children cannot be evaluated or expressed solely in terms of a certain decibel level There must be an interpretation of the total effect on the child's sensory, perceptual, intellectual, emotional and motor behavior In the study of these children it is also of vital importance that every effort be made to help the parents understand and accept their child's specific problems, and to help them through the various crises that occur at various stages in the child's development

*Behavioral Characteristics of Children with Communication Disorders*

The behavior of any child is the result of many factors, but the behavior pattern of the deaf child is basically the result of his sensory deprivation and its effect on his ability to learn, think, and communicate

*The Deaf Child* The behavior of the deaf child, by the age of 2, is very different from that of the child with normal hearing When the primary distance sense of hearing is impaired or lost the remaining distance sense, vision, and the close senses, take on different roles The deaf child is especially alert to any visible movements in his environment and he is very sensitive to vibrations Unable to hear and speak, he communicates and makes his wants known by using gestures and pantomime, because he has good understanding and inner language Failure to make himself understood may easily result in frustration and tantrums Unable to hear his own vocalizations, his voice becomes flat and toneless He does not improvise or play with sounds His motor development is good and he tends to be noisy and hyperactive His hyperactivity, however, has the purpose of bringing him into close contact with his environment with all of his senses other than hearing The deaf child is intelligent, friendly and cooperative, especially in the preschool age He is generally consistent in his day-to day behavior and not too difficult to manage in the home, provided the parents are accepting, affectionate, understanding and intelligent If he is not totally deaf, he will respond to sound stimuli, like a normal child, when the intensity reaches his threshold of hearing He responds more commonly to loud sounds of the lower frequencies rather than to the higher frequencies, i e over 1000 cycles per second This pattern of behavior is very characteristic of the congenitally deaf child It may well be used in the differential diagnosis of children with communication disorders as a frame of reference for those who are presumed to be deaf

*The Brain Injured Child* Although "brain injured" is vague, unscientific term it is used here for want of a better designation for those children who cannot as yet be conveniently diagnosed as having any specific type of brain damage or neurologic deficit The isolated or sometimes irregular

difficulties that they exhibit in the development of verbal or other forms of language, nevertheless appear to be the effects, or subtle expressions, of minimal and probably diffuse dysfunction within the central nervous system

In the study of these children, one is impressed by the multiplicity of the effects of so called brain injury, from whatever cause, in terms of disorganization or disunity of function, and unpredictable responses to sensory stimuli, be they auditory, visual, tactile, kinesthetic, or even painful The variety of manifestations depends of course, on the site, severity, and time of the brain damage In this context the brain injured child is one whose central nervous system was damaged as a result of anoxia, trauma, toxins or infections, before, during, or in fact any time after birth The damage may have been minimal or severe, focal or diffuse, cortical or subcortical, or in the brain stem There is now important experimental evidence to support the clinical suspicion that minimal lesions in the midbrain may be of major significance in these children

The brain injured child is different in every respect from the deaf child He may have disturbances of perception, concept formation, language and emotional control which are all reflected in his behavior These disabilities may or may not be associated with the type of motor involvement seen in cerebral palsy He is distractible, hyperactive, and inconsistent His hyperactivity is not as purposeful as that of the hyperactive deaf child His emotional behavior is often unstable and exaggerated He may be uninhibited fearless, happy and affectionate He may be given to violent uncontrollable outbursts of temper He is often destructive without anger He may be meticulous and pay unusual attention to minute details At the preschool age he may have a tendency to persist in repeating an activity once begun, long after it has ceased to have meaning or purpose, this characteristic is called perseveration He is difficult to understand and to manage in the home because he is so unpredictable and disorganized

Although he may be presented as a retarded child or a behavior problem, the brain injured child's disturbed language development raises the question of whether or not his hearing is normal His responses to sound are inconsistent and at *times remarkable He* may *not* respond *to* pure *tones* presented in a free field at intensities approaching the threshold of pain, and yet a moment later he may definitely respond to a whispered voice or the squeak of a toy rubber mouse, depending upon his attention at that moment

*The Autistic Child* The autistic child is very strange He may be confused with the feeble minded, or, because of his muteness and inadequate responses to sound, with the deaf The behavioral characteristics of this severe form of psychic disturbance were first described by Kanner in 1943 and called infantile autism This type of child is withdrawn, noncommunicative, self-sufficient, happiest when left alone, meticulous in details, adverse to changes in routine, and interested primarily in objects

and not in people, not even his parents He treats people as objects, shows no affection, and will rarely look anyone in the face These children are intelligent but their "cognitive potentialities are masked by their basic disorder", which has been defined as a "disability to relate themselves in the ordinary way to people and situations from the beginning of life" Their behavior is governed by an anxious obsessive desire for the maintenance of sameness that nobody but the child may disrupt He is given to panic tantrums followed by some sudden calm. His face is placid but occasionally he may be found smiling, humming or singing to himself Hebephrenic and catatonic types of schizophrenic behavior may also be observed in the most seriously disturbed children The hebephrenic type is characterized by curious mannerisms, gesticulations, tic-like movements, and unexpected spells of laughing and crying Compulsive and repetitive actions are common The catatonic type is more generally tense, rigid, and contrary Awkward positions may be maintained for long periods The autistic child pays no attention to his surroundings He may not respond even to a painful stimulus He may appear to be in a stupor Here again it is the lack of speech and the absent or inadequate response to sound which may lead to a suspicion of deafness The child's characteristic pattern of behavior, however, provides a clue to the basic disability An accurate assessment of the hearing capacity is most difficult because of the child's self-imposed isolation and lack of normal response to any stimulus The pathogenesis of this condition is unknown Although some of these children may remain mute, the majority apparently have normal hearing This may not be established until they are 8 to 10 years of age

*The Mentally Retarded Child.* The mentally retarded child performs at the level of his intellectual or mental maturation He is retarded in all phases of development, including language and perception He is unable to take care of himself His social, learning, and mental ages do not relate favorably to his chronological age The educability of retarded children can be determined by specific tests which are best applied by experienced psychologists who may interpret the results in terms of each child's intellectual potential If there is a suspicion of a hearing impairment the type of hearing test employed must be in keeping with the mental age of the patient An early diagnosis of mental retardation has been one of the most common, and tragic, diagnostic errors in children who were later discovered to have severe, abrupt, high tone sensorineural hearing impairments and poor discrimination abilities for speech In many cases they were later found to have normal intelligence A complete multidisciplinary evaluation therefore of all of these children is the absolute minimal requirement for a diagnosis and plan for future management

CHAIRMAN Thank you, Dr McHugh, for a most interesting and instructive film Does any member of the Conference wish to comment on it?

HAWKE I think that Dr McHugh has included the differential diagnoses which I think are most important Among the major problems, *the retarded child* should not be particularly difficult to diagnose because, with a good developmental history and observation of the child, you can usually assess, within broad limits, his intellectual capacity The point to remember is, of course, the possibility of the dual handicap,—some children may be both retarded and deaf This combination occurs in the rubella group

The most confusing, I think, is *the autistic group* We do not see them until they have matured beyond the stage of the primitive reflex responses to sound They do not respond in any way to sound in many of the tests for auditory capacity, and thus they appear to be deaf However, the differential diagnosis can be made reasonably well if you suspect this condition The concept of autism is the most important thing If one suspects it, then the history of early development, as obtained from the family, is pretty clear The child fails to relate to the world about him but lives in a world of his own In your own office you can see this isolation The child remains apart from you and the parents and everybody else The final diagnosis can often be made by placing a child in a group situation I like to have the child attend a nursery school and observe in detail the relationships which develop or which fail to develop rather than to make a definitive diagnosis at an office visit

*The organic problems* are very difficult One variety is the hyperactive, restless, *hyperkinetic* child who often does not focus and pay attention He is extremely difficult to test because of his distractibility and the variability of his responses This is, I think, the so called Strauss syndrome This often creates a false impression of deafness If you watch him carefully, however, you can find periods during which he will respond and the correct diagnosis can be made after prolonged observation Another variety is *true organic aphasia*, and perhaps we can come back to this topic later I will only say that at the age of 2 or 2 1/2 it would be very difficult to make this diagnosis The condition emerges gradually, and repeated evaluations are necessary These cases I believe usually represent a disturbance of development rather than an environmental disturbance By environmental disturbance I mean damage to the brain

McHUGH When this film was shown at the Neurological Institute one of the neurologists asked me afterwards if I realized that the child with the paper hat had organic brain disease I said that we had not identified any specific neurologic disease "Well", he said, "he is definitely an epileptic" Seven years later this child had his first seizure I was very much impressed

W HARDY In my medical institution we have a large group of what might be called iconoclastic psychiatrists, to the extent that the head of the department is quite as apt to be a specialist in cerebral circulation or

neurophysiology or neurochemistry as he is to be a classical psychiatrist From conversation with these colleagues I gather that the chances are pretty good that ultimately the so-called "psychic disturbances" described in the textbooks will ultimately be understood to depend on neurochemical imbalances of one sort or another With this in mind I would question whether our distinction between organic vs behavior defects is really a valid, legitimate, and useful dichotomy For example, current information tells us that a lack of or imbalance in certain proteins and amino acids not only interferes with conduction in primary projection systems such as the auditory system but also interferes very seriously with the functions of memory, retention and recall, all of which enter fundamentally into the behavior patterns of the children

DAVIS I suppose, Dr Hardy, a practical question would be, "Does this concept of a neuro-chemical deficiency lead us to a different kind of treatment such as by diet or by pharmacological means, or is, perhaps, the behavioral approach through training still the best thing that we can do?"

M HARDY I am very suspicious of neat, clean categories and I think that one of the greatest favors that this group could do is to put a stop to our labeling and simply recognize on the one hand a group of high-risk babies who are in trouble and on the other hand a group of professional people who are prepared to study and observe them from the medical, the paramedical, and educational points of view By such study we shall make progress in helping the specific child and also in gaining more general insight

We have too many cubby-holes that got created too early, and we do our best to shove the kids with problems into them according to neat descriptions as if the kids were static Actually they continue to grow, to mature, to suffer deprivation and a variety of other conditions both positive and negative They will need reassessment at regular intervals so that we can readjust our concepts, our techniques, and our methods of management

McHUGH I agree entirely with Dr Hardy, as she knows It would be a misconception to think that I am one of those who will pigeonhole kiddies

M HARDY God bless you!

CHAIRMAN Dr Barr, will you give us your comments as an otologist on these problems of differential diagnosis?

BARR I have seen several children with malformed ears This is the easiest diagnosis in the world, but I recall one of these children who had

been seen by many doctors They were very nice to the mother, telling her how they later on would help the child to a plastic surgeon but no one mentioned the possibility of a hearing loss The need for plastic surgery is so obvious that the doctors often forget that there might also be a hearing problem

DAVIS In other words, plastic surgery is easier than the education and rehabilitation of a deaf child

McHUGH The study group for language and hearing disorders at the Montreal Children s Hospital employs a battery of disciplines They review every child The representatives meet every week and review not only their technical reports but they attempt to come up with a program of what we can do to help the child and the parents more often than they come up with a definite diagnosis

I too want to emphasize once more the dangers of an unqualified early label for these kiddies We have had some very sad experiences of this sort Once a child is labeled as a deaf child or is placed in some other category it tends to make everyone sit back and say, "Well, that's all there is to it", as if nothing else were ever going to happen I think of a case in point, a child who was referred to us last week, who had been considered to be deaf or brain injured or aphasic or schizophrenic When this case was reviewed thoroughly it was clearly a child deaf from birth but with a very clear history of psychosis However, at the age of 8 years she had, within the last 8 months, come out of this infantile depression or schizophrenia or whatever we might call it She no longer presented at all the same problem that she had 8 months previously This is a story of a multiple difficulty and of the difficulty of assessing which is the major handicap

WHETNALL To illustrate the damage that can be done by labeling too early, I have recently had occasion to examine eight children who had been diagnosed as aphasic In my opinion three of them are children who were late developers They matured late in talking but by strictly leaving them alone, i e no treatment except to encourage the mother to talk to the child, they are now talking One is 4 1/2, one is 4, and the other is not quite 4 Another of the children is mentally defective with an IQ of 52 Another child is severely deaf with a 90 dB hearing level (ISO), combined with cerebral palsy Another child was premature, less than 3 pounds at birth, who presents general problems of development The last child is both deaf and very retarded

CHAIRMAN This illustrates some of the complications and some of the confusions

neurophysiology or neurochemistry as he is to be a classical psychiatrist. From conversation with these colleagues I gather that the chances are pretty good that ultimately the so-called "psychic disturbances" described in the textbooks will ultimately be understood to depend on neurochemical imbalances of one sort or another. With this in mind I would question whether our distinction between organic vs behavior defects is really a valid, legitimate, and useful dichotomy. For example, current information tells us that a lack of or imbalance in certain proteins and amino acids not only interferes with conduction in primary projection systems such as the auditory system but also interferes very seriously with the functions of memory, retention and recall, all of which enter fundamentally into the behavior patterns of the children.

DAVIS: I suppose, Dr. Hardy, a practical question would be, "Does this concept of a neuro-chemical deficiency lead us to a different kind of treatment such as by diet or by pharmacological means, or is, perhaps, the behavioral approach through training still the best thing that we can do?"

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# IV. DEFINITIVE TESTS OF HEARING

## A. Peepshow

CHAIRMAN (afterthought) We have now reviewed the questions of etiology of hearing impairments in very young children We have reviewed the possible screening tests for such impairments including prenatal as well as neonatal testing We have come to the conclusion that the results of neonatal testing are of uncertain value in that some infants are missed who should be detected while many who are detected turn out later to have no defect We have developed as an alternative the concept of the High-Risk Register, based on our knowledge of the probable etiology of such impairments of hearing

We have discussed not only the tests which are applicable to the newborn infant but also simple tests which may be used effectively during the period from the sixth month to the third or fourth year We have also considered the problems of differential diagnosis that so often confront us in the examination and assessment of a young child who does not learn to talk at the usual age

We are now faced with a choice of strategy, whether to turn at once to the more elaborate tests, based upon electrical responses of one kind or another, which may give more definite information even in the early months or years, or whether to consider first the more familiar types of test, notably play audiometry and the peepshow, which merge ultimately into the classical pure tone audiometry which constitutes the definitive test for adults and for older children

In this written version of the conference we shall rearrange the actual sequence slightly and we shall devote a few more pages to "play audiometry" than were actually spoken in the conference We shall then return, at a slightly more technical level, to the various electrical tests, old and new

I will ask Dr Dix if she will tell us the present status of the "peepshow" and perhaps whether I was correct, in the agenda, in calling this test an operant conditioned reflex

### *The Peepshow Test*

DIX The Peepshow, as *first described (Dix & Hallpike, 1947)* was *designed* to overcome the great practical difficulties of obtaining rapidly a reliable pure tone audiogram in young children with speech defects thought to be

due to deafness. The ages of the children were within the period three to six years, at which an accurate assessment of hearing capacity was urgently needed for the satisfactory prescription of the children's educational management

In 1952 (Dix & Hallpike) a more comprehensive paper was published, giving results in a series of 260 cases, and in the course of this work the reliability of the findings was confirmed by a detailed follow-up Since then the method has continued to give satisfactory results Furthermore, the apparatus itself has been improved and simplified without, however, any departure from the very simple principles of construction and performance originally laid down

The principles of the procedure are shown in Fig 1 It is designed to avoid the two great difficulties which make conventional pure-tone audiometry impossible with young deaf children In the first place, otherwise meaningless pure tones are given an arresting significance In the second place, the need for any explanation of the nature of listening and hearing is completely avoided The test procedure depends upon the conditioned response of the child to a series of short pure-tone stimuli delivered from a loud-speaker The pure-tone stimuli are synchronized with the flashes of a signal lamp

The child sits in front of the box, in which are displayed a series of attractive pictures In order to see these pictures, the child must first illuminate them by the pressing of a button Thereafter, he inspects them through a viewing aperture This press-button mechanism works only when the synchronized light-and-sound stimuli are being delivered

The stimulation mechanism is under the control of a tester, who observes the child closely from behind a screen The pure-tone stimuli are variable in frequency in fixed octave intervals from 250 to 4000 c p s The intensity is calibrated in decibels above the threshold of hearing of a normal subject with his head in the position of the child During the test, an instructor is seated by the child's side and performs the important functions of controlling him, encouraging him to co-operate in the test, and of changing the picture by the operation of a simple mechanism when necessary

The test begins with the instructor focusing the child's attention on the viewing aperture When the tester observes that this has been accomplished he applies the double signal, the signal lamp flashes and the loudspeaker emits synchronized impulses of sound The instructor at once presses the button illuminates the picture, and encourages the child to inspect it, which he usually does with every appearance of interest When this has been accomplished the tester withdraws the signal and the picture disappears

After the demonstration has been repeated two or three times, the child soon learns to press the button and illuminate the picture when, and only when the double signal has been given As soon as this reflex has been est[illegible] next step is to eliminate the light signal in front of the

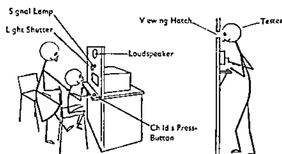



FIG. IV. 1

box. This the instructor does by closing a simple shutter over the light. The test is then continued using the interrupted sound stimuli only, and thus becomes a test of hearing.

In children without hearing this elimination of the light signal makes further response impossible. On the other hand, in children with sufficient hearing to detect the sound signal the elimination of the synchronized light signal makes no difference to their response and they continue to react to the sound signal alone. The intensity of the sound signal is then reduced progressively until a threshold reading is obtained. The test frequency is then changed and the final result is a conventional audiogram. This, with a co-operative child, may take less than five minutes.

The tester's part of the apparatus is simple. It consists of a pure tone oscillator giving frequencies fixed in octaves from 250 to 4000 c.p.s. In it is incorporated an attenuator giving outputs from 10 to 90 decibels above threshold. The interruption of the tone and the light is carried out electronically. The frequency of the sound pulses can be varied, thus lessening artefacts due to standing waves.

Great importance is attached to the management of the children to ensure successful testing by the Peepshow. They are always seen by appointment. Precautions are taken that they are not examined in a peevish or fretful state after a long journey or early start. Their intelligence and general suitability for the test is assessed at a preliminary interview. Successful Peepshow audiograms have been carried out at Queen Square in children to a lower age limit of two years.

Since the Peepshow was first introduced a good many papers on the subject have appeared. Some of these have been favorable to the method, others unfavorable. In determining this latter trend of opinion there seems no doubt that a part is played by aberrations of technique. Most of these seem to arise from a certain human insistence upon introducing improvements of one kind or another, and in a number of equipments that have been examined it can only be said that these improvements are of a kind that obviously excludes any possibility of success. Perhaps no more need be said on this aspect of the matter, except to stress that the Peepshow,

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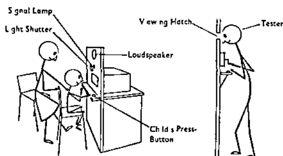



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as a machine, is one of some complexity Its design and maintenance are not easy matters for most clinics, and unless both are entirely satisfactory disappointment is inevitable Certainly in most clinics in our country other methods are used and it is often asked nowadays what place the Peepshow can be said to occupy in the armamentarium of the modern paedo audiologist Often, too, it is suggested that it is quite unnecessary and many authorities would say that they get results as good with much simpler methods of play audiology

These views are no doubt very well founded, based as they are on the very favorable conditions which are now available in most paedo audiological clinics Here, ample time and repeated test sessions are possible, in the course of which play-audiometry techniques give reliable results

All this, of course, is to the good and reflects much credit on those who are responsible for the large scale organization of this work At the same time, it cannot yet be said that these excellent conditions exist everywhere In some parts of the world it is still necessary to see children, as they were seen at Queen Square, brought from a long distance with little opportunity for repeated tests In this situation the need is still for a test procedure of the highest possible efficiency which will give a reliable result in the course of a single and short examination In this situation the Peepshow may still be of value

This opinion is not ventured at random, and Dr Lang of Budapest (Lang *et al*, 1963) has reported that in her clinics there, and elsewhere in Hungary the practical matter of hearing tests in these young children is still one in which there are few facilities for repeated observations, and that in her hands the Peepshow has proved of the greatest practical value Trenque (1964) and others have had similar experiences

## *Discussion following Dr. Dix's discussion on the Peepshow*

CHAIRMAN Dr Statten, I believe you have had some personal experience with the "peepshow" method Would you like to add a comment?

STATTEN I have very little to add to Dr Dix's excellent presentation At the Hospital for Sick Children in Toronto the late Dr Wishart used some of Dr Dix's ideas He developed a peepshow in the form of a doll's house The idea is the same A standard pure-tone audiometer is operated by the observers The upper window of the house in front has a loudspeaker and beside the front door is a doorbell that the child operates in response to sound

(Dr Statten illustrated the equipment by showing lantern slides )

A 16 mm movie projector shows pictures on a ground glass screen in the end of the doorway The child presses the button when the sound is on and thus starts the moving picture He learns this very quickly by demonstration and he is interested in what he sees

We have been using this form of the peepshow for 15 years at the Hospital for Sick Children We believe that any hard of hearing child who is actually normal in other respects should be able to perform this test satisfactorily between the ages of two and three After he becomes accustomed to it using the loudspeaker we then use earphones to determine audiograms for the two ears individually and we can use bone conduction vibrators as well In the acoustic field we can compare the improvement of pure-tone thresholds that is obtained by use of a hearing aid

We find this a very simple and enjoyable experience both for the operator and for the child We realize that it has limitations which depend among other things, on the intelligence of the child

CHAIRMAN Thank you, Dr Dix, for an extremely valuable and informative description of the "Peepshow" and its particular place in the armamentarium of the otologist and audiologist Let us now complete the picture with a brief discussion of "play audiometry" by Dr Barr

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## B. PLAY AUDIOMETRY

BARR Play audiometry comprises two equally important phases The first phase consists of *learning* the second of *threshold determination* The success of the entire examination depends on the learning technique

*Our procedure is as follows* The child is placed at a small table beside the audiometer where the examiner can keep an eye on him all the time The tone interrupter is manipulated with the right hand, and the play instruction is given with the left

*The play instruments are placed on the table in front of the child* They are colorful, simple educational toys, toys that can be used in what may be called a sort of "series game"

The procedure is started by presenting a tone that the child may be expected to hear on the basis of preliminary examinations At the same time that the tone is transmitted, the child is shown with a quick, sharp motion how to move the toy The movement must be made *demonstratively* so that it catches the child's attention When the next tone comes the demonstration is repeated, continuing thus until the child appears to have understood the procedure The child soon wants to carry out the little demonstration himself The moving of the toy becomes a "reward" for perception of the tone, while the "penalty" of being prevented from per-



Fig. IV. 2. Test arrangements for play audiometry.

forming a pleasant action makes it clear to the child that he has not followed the "rules of the game" if he does not wait until he hears the tone. Accordingly, the learning is based on traditional trial and error principles.

*Play audiometry* succeeds in approximately two-thirds of cases for children between two and one-half and three years depending on the child himself, the time available, and the skill of the examiner. The threshold determinations vary more for children than is usual for adults on retesting the same or the following day. Play audiograms should therefore always be based on at least two tests in which the divergence for each frequency does not exceed 5 dB. Follow-up examinations ten to twelve years after the primary examination have shown good agreement with such first tests. When play audiograms are not consistent at the primary examinations, the play audiograms at follow-up may show considerably lower threshold values than those obtained at the primary examination.

Comparison between threshold determinations obtained with *electrodermal response and play audiometry has shown close agreement and no statistically significant difference* was demonstrated for seventy children examined with both methods. However, play audiometry can be used with success only exceptionally with children under two and one-half years while electrodermal audiometry is often possible at a younger age although it too fails in perhaps half of the cases.

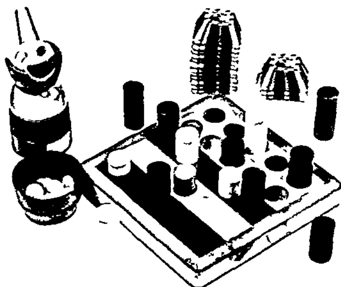


Fig IV 3 Educational toys that proved suitable for play audiometry

The question whether or not either one or the other of the methods is preferable as a threshold determination test might be as follows In the cases where both methods are applicable, the choice is evident The equipment needed for play audiometry is less difficult to operate than that necessary for electrodermal audiometry, and the method is less time-consuming and requires less personnel than electrodermal audiometry. It is based on a pleasant experience while electrodermal audiometry is unpleasant Play audiometry should always be attempted first, assuming the child is old enough so that successful results may reasonably be anticipated Electrodermal audiometry might well be reserved solely for the cases in which play audiometry has failed, or where because of the child's age it might be expected to fail

Chairman Thank you, Dr Barr I know that at this point we could go into considerable debate as to the relative merits of play audiometry and the peepshow I think many of the precautions you have both mentioned about the handling of the patient are extremely important, and perhaps they relieve us of the necessity of going into further details about classical play audiometry The two methods, as I see them, are based on the same general concepts, and both Dr Dix and Dr Barr have described some very helpful refinements of method

Lowell Our play audiometry is very similar to that in use in other laboratories We find that it is useful with cooperative children as young

as two years. We conducted a study of test-retest reliability on a group of children who had been seen earlier at the Clinic The average age of a group of 21 children was three years, six months We retested them at an average age of six years, eleven months, when it would presumably be possible to obtain more reliable data The comparison of the test-retest threshold shows differences that are well within the standard error of clinical audiometry.

Another way of evaluating this technique is to estimate its validity against the criterion of normal hearing Tests of a group of 21 children with normal hearing between the ages of two years, six months and three years, four months were conducted Again the values were so close to the predicted values that one can reasonably assume that this technique is valid with children of this age range The technique has been photographed on 16 mm color motion picture film and is available for training purposes

W Hardy I find myself allergic to pure tones

Statten We use voice tests in other situations but we haven't tried to adapt the peepshow to speech audiometry

W. Hardy You would get much closer to the point of it all with speech

Statten We've been satisfied with the results we have been getting in the normal deaf child

Whetnall I would like to emphasize each word of Dr Hardy's remarks I can't see the point in testing with pure-tones Over and over again I have found no relation between the pure-tone audiogram and the speech which the child can ultimately develop There are learning effects that occur with pure tones as well as for the more complicated speech sounds We should start testing the hearing for speech because it is how they are going to hear speech and how they are going to respond to speech that we want to know

Barr Pure-tone audiometry can be used in two ways We need a hearing test when we start an educational program for the deaf child, an audiogram for reference later We also need audiograms for grown-ups If I am a surgeon and perform an operation I want to assess the result The basis of this assessment is the pure-tone audiogram

Let's go back to young children A child may be brought to a skilled audiometrist but one who doesn't know anything about children On the other hand a child may be brought to a teacher or doctor accustomed to working with kids They know how to handle children but they don't know how to handle the apparatus If we are to know ten years later whether there [illegible] a change in threshold as a result of training or the use of

hearing aids we must rely on pure tone audiograms I believe that play audiometry gives the best basis for such comparisons later, but we need specially trained people who know how to handle children I use kindergarten teachers and they are also trained teachers for the deaf whom I train in audiometry We take many audiograms I usually say that one audiogram is worse than nothing We must have at least two and these two should agree at each frequency within five dB It is quite possible to get such audiograms although it may take you ten days to do it, but if we are going to compare our results of training or perform surgery we must be able to rely on these first audiograms

A EWING Dr Ian Taylor, my successor, in his new book (1964) reports differences in responses of children during sleep, depending upon whether the acoustic stimuli are familiar to them It has seemed to us very clearly that in all subjective audiometry, whether speech audiometry or pure tone audiometry, there is a learning process and the results depend on how far the sound is already meaningful or can be made meaningful

Also the state of attention of the child is extremely important, particularly in the type of tests we have described We employ deliberate techniques to render the child responsive to sound With an infant, one worker provides visual stimulation for the child at the beginning of the test but this stimulation is not continued at the time the sound stimulus is produced The other stimulation is removed as far as possible because a child who may be incapable of attending simultaneously to an auditory stimulus near threshold and to a visual display might be too much interested in the latter and thereby prevented from responding to the auditory stimulus

I agree with Dr Barr that we are constantly required to produce audiograms for ENT consultants and one of our aims is to be able to determine them accurately at an early age I have here the results of pure-tone tests of 13 children aged about two years and three months, determined by play audiometry I believe the results are reliable We shall publish the data later after we have acquired much more of it It is a regular routine with us to make pure-tone tests at the earliest possible age and to *teach* the child (there is no other suitable word) to respond to sound

Dr Kendall, back in the early 50 s was producing material to use as the basis for speech audiometry with very young children The trend here is to use recorded speech whenever possible rather than monitored speech My wife has produced some material which combines activity for a child of say 18 months or 30 months so that we may begin to learn how far the child is beginning to understand speech

On the other hand we are still using the pitchpipes that were originated in the Neumann Polyclinic, Vienna These are made with organ reeds They are suitable for field testing and may be used with some children at an earlier age than we can use pure-tone audiometry It is a sort of transition You can make a game out of it with the child, first letting him see

you blow the pipe and then making a transition from this to the audiometer receiver which is put on his ear while he is engrossed in the game and doesn't know whether the receiver is there or not It is an interesting question how far we must regard these children as *practiced patients* for whom it is now an acquired skill to respond to sound at something like *threshold intensity*

DOWNS: Classical "play audiometry" offers the clinician a variety of techniques to elicit a voluntary response in young children Because the essence of the testing situation with young children is the personal relationship between the tester and the child, the clinician should have available to him the kinds of procedures that fit any tester-child relationship My *own experience has led me to prefer to have as little equipment between* myself and the child as possible, and to keep that equipment extremely simple Following are suggestions for the kinds of techniques that are useful

*For the child 2 to 3 years old*

Simple, unadorned blocks, 2″ by 2″, which he is taught to pile up when *he hears a tone*

Plain marbles which he drops into a box or a bottle when he hears the tone

*For the child 3 to 5 years old*

Pegs and peg board, with an animal or car in the middle of the board, he is taught to "build a fence" or a "garage" when the tone is presented

*A fenced enclosure with small farm animals to put into the enclosure* when the tone is presented

A number of small cars or airplanes to put into an enclosure when the tone is presented

*One should remember that the above ages apply to mental age rather* than to chronological age Therefore, these techniques are useful also for *the mentally retarded child whose mental age falls in the above categories*

GOLDSTEIN (in absentia): Testing of infants with pure tones is as useful and as valid as testing with speech if we set as a goal the determination of auditory sensitivity and the probable effects of reduced auditory sensitivity on the understanding of speech

It is difficult to conceive of speech being significantly more meaningful th*an pure tones to a newborn child or to an infant of a few weeks* Similarly an unschooled child with a profound impairment of sensitivity could hardly f[ind] speech stimuli significantly more stimulating than speech to which [he had nev]er attached meaning

[...] ch stimuli to determine auditory sensitivity can in fact [...] misdiagnoses of aphasia or other central auditory

disorders to which Dr Whetnall referred Good sensitivity to the low frequency components of speech can obscure a high frequency loss In this situation reactions to speech can be elicited close to normal thresholds If the high frequency loss is not detected by pure tone audiometry then the failure of a child to develop normal understanding of speech may easily be misascribed to some central auditory defect

High frequency sensitivity can be tested with sounds other than pure tones Pure tones however, are usually easier to calibrate control and specify Many earlier investigators claimed that noisemakers and natural environmental stimuli were more effective than pure tones of equivalent sound level in eliciting responses from infants and young children Most of the older pure tone audiometers had very slow rise times whereas the noisemakers usually produced more abrupt stimuli Although the proof is still equivocal it is fairly certain that pure tones with rapid rise times although not short enough to produce clicks are more likely than pure tones with long rise times to elicit responses

I fail to see how one audiogram is worse than none The implication of this statement is that in serial audiograms the first audiogram bears no relation to subsequent audiograms This does not seem to be the experience of skilled clinicians The zone of uncertainty may diminish in subsequent tests but not necessarily the audiologists estimate of the hearing level (Goldstein *Folia Foniatrica* 1962) If the threshold measures by pure tones are uncertain the threshold measure by speech is often just as uncertain

Davis I wish to emphasize once more the importance of the proper degree of arousal or attention or motivation of the subject This point has appeared in the discussion of practically every test from Dr Wedenberg s awakening test and the Ewing method directly on through both play audiometry and the peepshow The Ewing distraction method and the peepshow were both designed specifically to obtain the proper degree of attention interest and cooperation We shall see that the problem of the degree of arousal appears again in the electrodermal and electroencephalic methods The condition and attitude of the subject must never be forgotten

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## C Auditory Reaction Time and Other Tests

Chairman Dr Rapin has developed another modification of audiometry for young children that employs quite a different criterion namely the

latency of the child's response This is an interesting modification Dr. Rapin, will you tell us about it?

## *Auditory Reaction Time as a Test of Hearing in Children*

"Auditory reaction time as a test of hearing in children" is a description of work carried out by I Rapin, L D Costa and I J Mandel of the Saul R Korey Department of Neurology *and* Bela Schick Department of Pediatrics of the Albert Einstein College *of* Medicine, New York, New York This work was supported by grants NB 2503, NB 3356 and 2TI NB 5325 from the National Institute of Neurological Diseases and Blindness, United States Public Health Service

RAPIN One of the chief difficulties in interpreting audiograms of children suspected of severe hearing loss is to evaluate how well the children are motivated to respond Operant conditioning techniques like the peepshow have been introduced with the hopes of keeping motivation high They are said to be quite effective in this respect Nevertheless, the possibility that lack of motivation accounts for some children's failure to respond still exists and might not be detected It would seem desirable to assess motivation in the audiometric testing situation independently of the procedures designed to test hearing itself Reaction time, that is the time elapsed between presentation of a stimulus and the subject's response, pressing on a key for instance, can be used to evaluate motivation since rapid responses occur only in motivated individuals To maintain motivation at a high level, rapid responses can be reinforced

Non-verbal children can be expected to respond to visual stimuli like normal children of the same age unless lack of motivation or severe central nervous system damage is present A child who responds rapidly and consistently to suprathreshold visual stimuli and *not at all* to auditory stimuli is probably not perceiving the sound If a child responds fast to light and slowly to sound, it is possible that he is motivated but that the sound is near his threshold since reaction time is known to increase markedly when stimulus intensity approaches threshold (Chocholle, 1945)

We undertook two experiments to study auditory and visual reaction time in normal children and in children attending St Joseph's School for the Deaf in New York City (Costa *et al*, 1964) In the first experiment we studied reaction time to a light flash, to white noise at 70 dB above threshold of normal hearing, and to paired light and sound in 28 normal children aged six and one-half to ten years, and in 50 children of similar age from St Joseph's School, some of whom were peripherally deaf, while others were suspected of mixed deficits including brain damage Ten visual stimuli were followed by ten auditory ones, then light, sound, and paired light and [illegible] were presented in random order In the normal children, we found [illegible] reaction time was shorter than visual reaction time and that reaction [illegible] paired visual and auditory stimulation was shorter

still We found a significant improvement in reaction time over five successive experimental sessions, and with reinforcement consisting of giving the child an M & M candy for responses below the median of the previous day

Visual reaction time in the children from St Joseph was similar to visual reaction time in the normal children, and showed the same improvement with reinforcement and on repeated days Pairing of visual and auditory stimuli did not shorten reaction time except in two children with language deficits unassociated with a hearing loss Among the 50 children from the school, six responded to sound alone from the onset and seven started to respond during the course of the experiment (Table I) We were unable to show that it was pairing of auditory stimuli with light which was responsible for the learning which occurred in those seven children

We then performed a second experiment to study changes in reaction time as stimulus intensity was lowered toward threshold Three normal children were tested with light of constant intensity and with white noise at 50 40 and 30 dB above threshold of normal hearing On subsequent days the intensities were lowered in 10 dB steps until the child stopped responding to the lowest intensity One child did not respond below 20 dB, the two others were still responding at 10 dB Because testing was carried out in a noisy environment, we did not attempt to see how much closer to threshold responses could be obtained The striking increase in reaction time at 30 dB for the child who did not respond at 10 dB, and at 20 dB for the child still responding at 10 dB can readily be seen in Figure 4

Twelve children from St Joseph's School who had responded to sound at some time during the five sessions of the first experiment, and ten children who had not responded to sound alone were then tested as above, but the intensities used on the first day were 70, 60 and 50 dB above threshold of normal hearing (Table I) Again, if the child responded to

TABLE I *Response to White Noise of 50 Non verbal Children*

| Experiment 1 Response at 70 db | | Experiment 2 Threshold determination | |
|---|---|---|---|
| Responded immediately | 6 | Had normal threshold | 2 |
| Started to respond during the course of the experiment | 7 | Had threshold between 50 and 70 db | 9 |
| | | Did not respond at 70 db | 1 |
| | | Not retested | 1 |
| Total | 13 | Total | 13 |
| Did not respond to sound alone | 37 | Had threshold between 50 and 60 db | 2 |
| | | Did not respond at 70 db | 10 |
| | | Not retested | 25 |
| Total | 37 | Total | 37 |

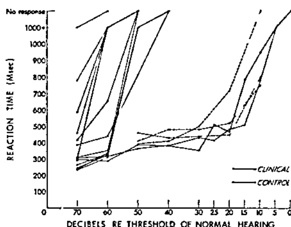



FIG IV 4 Auditory reaction time as a function of stimulus intensity in three normal children and in 13 non verbal children (Reproduced from Costa *et al* 1964 with the permission of the editor)

the lowest intensity these intensities were lowered in 10 dB steps until the child failed to respond to the lowest one over three successive days Short responses to light and all responses to sound were reinforced by one M & M candy Figure 4 shows that the children from the school without hearing loss responded as did the normals and that in both of them responses were obtained at 5 dB above threshold of normal hearing Reaction time increased abruptly at 15 dB in one and at 20 dB in the other child

Among the 12 clinical children who had responded to sound alone in the first experiment, one failed to respond in this experiment Of the 12 who had failed to respond in the first experiment, two started to respond during threshold estimation and they failed to respond to stimuli below 50 dB Figure 4 shows the pattern of responses of these 11 children, none of whom responded below 50 dB above threshold of normal hearing Again the slope of the curve of reaction time as a function of intensity increases rather sharply 10 to 20 dB above the intensity level to which the child fails to respond

The threshold estimated by this method was compared with thresholds obtained by conventional pure tone audiometry These comparisons are rather crude since our experiments were not performed in a soundproof room, and since we used white noise rather than pure tones We compared the threshold obtained in this experiment with the threshold of the best ear at 1000 cps Of 11 children who did not respond to white noise at 70 dB above threshold of normal hearing in this experiment, eight had pure tone thresholds for 1000 cps at 70 dB or higher, and three had hearing losses of 50 or 60 dB for 1000 cps In the 11 children who responded be- 70 and 50 dB, our threshold estimate fell within 10 dB of threshold a nine children and within 15 dB in the remaining two

W r in these experiments that reaction time to visual and

auditory stimuli intermixed, with reinforcement of fast responses to light and of all responses to sound, is a promising method to study auditory function in children, although this small study will need to be validated in a soundproof room using pure tones Children five years and above were able to cooperate An IQ in the dull range, and suspected or definite brain damage without motor handicaps did not interfere with testing Each child acted as his own control in evaluating his reaction time since the younger the child the longer his reaction time (Teichner, 1954), and since brain damage also prolongs it (Benton and Joynt, 1959)

There was no doubt that reinforcement kept motivation at a high, perhaps optimally high level The use of light as well as sound has two advantages Delivering some stimuli which are readily perceived and responded to prevents attention from flagging and keeps motivation high Secondly, the latency of responses to light gives definite indications concerning factors which might give an erroneous impression of deafness such as lack of motivation, distractibility, or brain damage In other words, a child who responds fast to light and slowly or not at all to sound probably has a specific deficit in the reception or perception of sound, not an intellectual, emotional, or organic cerebral deficit Slow or erratic responses to both light and sound would on the contrary suggest that problems other than deafness are present

Play audiometry, the peepshow method, and reaction time are similar in that they indicate whether auditory stimuli are being used to program behavior All three techniques are presented to the child as a game so that his cooperation and attention can usually be held long enough to obtain useful audiometric data The special advantage of reaction time is that it not only gives a yes or no answer about whether the sound is serving as a signal, but that it also indicates that threshold is being approached 10 20 dB before it is actually reached since reaction time was found to increase sharply at that point in all children

The equipment to measure reaction time can be very simple and inexpensive The experimenter can program the stimuli manually His switch starts the clock and turns on the stimulus simultaneously The subject's switch turns off both the stimulus and the clock The experimenter reads the reaction time off the clock, records it and delivers the reinforcement to the child The stimuli need be no more elaborate than a light bulb powered by a battery and a standard audiometer with an added switching device such that it will stay on until the subject turns it off by pressing on his key

In conclusion, reaction time to mixed auditory and visual stimuli seems to us a simple and promising technique to assess hearing in children Our small study will need to be validated in a clinical setting using pure tones Variables such as schedules of reinforcement, interval between trials and the manner of presentation of the stimuli will have to be studied to establish the most effective way of using this technique

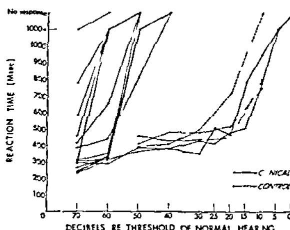



FIG. IV. 4. Auditory reaction time as a function of stimulus intensity in three normal children and in 13 non verbal children. (Reproduced from Costa *et al.* 1964 with the permission of the editor.)

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We conclude from these experiments that reaction time to visual and

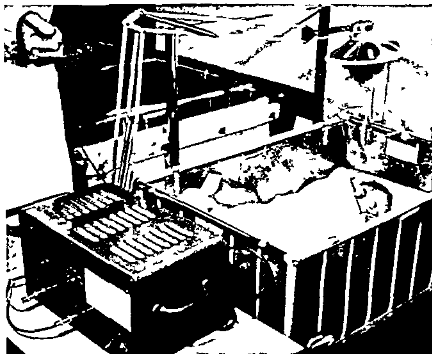


Fig IV 5 Arrangements for the auditory tests on newborn infants

utilization of electrical responses as the criteria of hearing One type of response is mediated through the autonomic nervous system and the electricity is generated in the skin Another variety involves voluntary muscles and their electrical action potentials as recorded from the head or neck The final class of electrical tests is based on the ongoing electrical activity of the brain itself (the electroencephalogram) or on specific evoked responses of the cerebral cortex

Dr William Hardy is a recognized authority on electrodermal audiometry, which is based on the so-called "skin galvanic reflex" or "psycho-galvanic reflex" He will summarize his present views on this subject for us

## *Some Considerations of Electrodermal Audiometry*

W Hardy There are two distinct electrodermal responses to acoustic stimuli We may employ either the Feré or the Tarchanoff "effect" The "Feré effect" refers to changes in the resistance of the skin and is measured by applying a voltage to the skin by electrodes and observing the change in current The "Tarchanoff effect" refers to changes in direct-current potential between the two electrodes in contact with the skin The equipment is almost the same for both Although the electrophysiologic principles involved are somewhat different, both procedures reveal an activity of the

### *The Awakening Test*

WEDENBERG. There are too many hearing tests for children. I propose only one, tone pulses at 3000 c/s, to awaken the child from light sleep. Adequate hearing for 3000 c/s predicts the ability to learn to speak normally, given normal intelligence, of course. Hearing impairments greater than the impairment at 3000 c/s are found in less than 1% of cases, and of course any suspected deafness can be tested further. This test is appropriate at any age. Uncooperative children can be tested thus even in late childhood. I would call this an awakening test. Movements of the eyelids, changing heart rate, and general bodily movements are the criteria.

In the awakening test I use a 3000 c/s tone, at 75 dB sound pressure level. The tone is presented intermittently, like a telegraph code, for one minute. I first ascertain that the stage of sleep is not too deep by stroking *the eyelash with a finger. If a reflex movement of the eyelid can be elicited,* a negative reaction to the tone, i.e. failure to wake within on minute, constitutes a valid result. (See Fig. 5.)

### *The Auropalpebral Reflex*

The auropalpebral reflex is useful for differential diagnosis. This reflex consists of a rapid and distinct closing of the eyelids in response to tones of high intensity under standard conditions. Frequencies from 500 to 4000 c/s may be used, at sound pressure levels between 105 and 115 dB re 0.0002 $\mu$bar. With a retrocochlear or a conductive deficit, it is impossible to elicit an auropalpebral reflex, but cases with cochlear impairment show positive auropalpebral reflexes, just as normals do.

One can essentially do an audiogram, in retrocochlear or conductive cases, by changing frequencies and raising the sound pressure level. In cochlear cases this is impossible because of the uncertainty introduced by recruitment.

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## D. ELECTRODERMAL AUDIOMETRY (EDA)

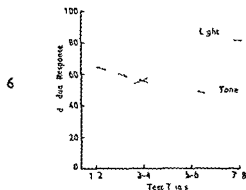
CHAIRMAN. I shall not attempt to generalize further about the nature of the reactions that are employed as criteria in the auditory testing of children. Let us turn at once to a set of tests which have in common the

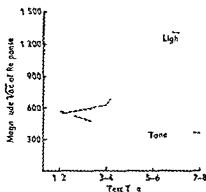
little clinical value as they cannot be employed with young deviant children with whom a test must be accomplished as quickly as possible in a difficult situation In effect this means that in the child our criteria must be sufficiently elastic, although definite, to meet the unexpected requirements and limitations of a given test situation As Grings and others have pointed out, conditioning is employed to sustain the subject's responses to sound Pediatric audiologists cannot well be limited by criteria for conditioning that have been developed with normal hearers in studies in experimental psychology Those test circumstances are neither comparable nor pertinent

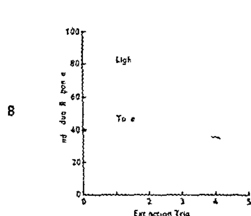
Another problem centers in the fact that the electrodermal response (EDR) is a nonspecific response It is well known that the EDR shows the same pattern not only to the test stimuli but also to many exogenous or endogenous stimuli that are quite unrelated to the objectives of the test So long as one obtains electrodermal responses to tones and can differentiate these from the responses to other stimuli, the test has fulfilled all reasonable requirements How to be certain of this differentiation, however, is often a difficult matter Various attempts have been undertaken, by Stewart, Hind, and others, to meet this problem by special instrumental controls or by judgments limited to sharply defined features of amplitude or latency of the responses

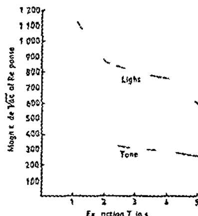
Although the data on normal adults can scarcely be related directly to the performance of children, deviant children in particular, it seemed important to know how frequently cooperative adults reveal chance responses under good test conditions By implication, an audiologist should heed the possibility of much higher rates of chance responses from small children who are apt to be something less than fully cooperative To this end we have studied the rate of occurrence of chance responses, as well as some other technical problems, by means of the Tarchanoff effect in EDA The occurrence of chance responses was usually 25 to 30%, depending on the exact criteria, and ranged from 0% to 60% Because of the large individual differences of the rate of chance responses, EDR 'threshold' limits need to be determined on the basis of each subject's own spontaneous activity, rather than in terms of rigid criteria for the definition of 'threshold"

LOWELL I would like to reinforce a distinction which I believe is important in discussing the use of electrodermal tests that is, the difference *between the use of this technique by audiologists and its use by research* psychologists studying learning I believe that in most of the uses of GSR (galvanic skin response) in audiology we are not following procedures that would allow us to describe this as conditioning A major goal of some psychologists is to determine whether conditioning has occurred, which requires a demonstration that the response to the conditioned stimulus after pairing with the unconditioned stimulus is different from what it was before paired stimulation It is also necessary to show that these changes are not due to fatigue, sensitization or adaptation

6 


 7


8 


 9


Figs IV 6 and IV 7 GSR conditioning of young deaf children Tone and light stimuli are interspersed in the conditioning trials

Figure IV 6 (left) shows percentage of positive GSR responses

Figure IV 7 (right) magnitude of responses

Figs IV 8 and IV 9 GSR conditioning in young deaf children Tone and light extinction trials are interspersed

Figure IV 8 (left) shows percentage of positive responses

Figure IV 9 (right) magnitude of responses

The task of the audiologist is to use the optimum conditions for sustaining the galvanic response to sound stimuli and to specify some criteria to define when a response has occurred This generally involves evaluation of latency and amplitude characteristics and some provision for detecting spontaneous responses

In our careful study of GSR *conditioning* of young deaf children in which tone and light were interspersed in the conditioning trial we find good evidence for conditioning to the light and little evidence for conditioning to the tone as shown in the Figures 6 and 7 The first indicates the percentage of the responses and the second the magnitude These are based on responses that were at least a 100 ohm change occurring within latency limits of 1 to 5 seconds

A similar pattern appears in extinction trials This experiment was car

ried out with a carefully counterbalanced design using adequate pseudo-conditioning controls Figures 8 and 9

While many interpretations of these results may be made, I think they raise very serious questions about the use of the notion of GSR *conditioning* with young deaf children

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## E ELECTROENCEPHALOGRAPHY (EEG)

CHAIRMAN Another major class of electrical responses that may be used as criteria for responses to auditory stimuli is the changes in the pattern of the electroencephalogram These do not represent voluntary responses

on the part of the subject and they do not require any training or conditioning, but they do require either an experienced observer or, as we shall see later, a computer to extract the relevant information from a welter of irrelevant ongoing electric activity. To open our discussion of electroencephalography I will ask Dr Bertrand to say a few words concerning the broad relations between impairments of hearing and the types or classifications of EEG

## *EEG Studies in a Deaf Population*

From the Speech and Hearing Center, Notre Dame Hospital Montreal, Director Doctor Fernand Montreuil This study was made possible through a Federal-Provincial Grant No 604-7 396.

The EEG classification and interpretation were done by Doctors Claude Gauthier and Normand Giard, neurologists, Notre Dame Hospital

BERTRAND In recent years, many papers have been published concerning the effects of evoked auditory potentials upon EEG recordings. The main purpose of these studies has been to obtain physiological data which should help us evaluate the degree of hearing impairment in young children as early as in the first days of life, especially in cases classified as a "high-risk group" Recent publications have shown the importance of detecting at the earliest age possible the presence of a hearing impairment in order to begin auditory training, even as early as six or nine months

Before starting the application of auditory evoked potentials on EEG pattern in relation to deafness, we wondered whether there was a classification of EEG's characteristic for a deaf population As we could not find any related exclusively to a deaf population, we proceeded to review EEG's of our own group

At the Speech and Hearing Center, of Notre-Dame Hospital, we have been recording EEG's on all the children referred for speech or hearing problems This has permitted us to have EEG's of a population of deaf children, whatever the etiology of deafness might be, rather than selective EEG's of deaf persons having a neurological reason for requiring such an examination

*Material* A review of 500 consecutive cases of deaf children has been made in regard to the EEG studies and clinical findings

All of these children had a complete investigation which included an examination by an otolaryngologist, a neurologist, a speech therapist, a pediatrician and a psychologist An investigation of the family background [illegible] was made with the aid of a questionnaire and a social worker

[illegible] the audiometric evaluation and the EEG recordings, which [illegible], other tests such as X-rays, tests of biochemical or [illegible] were done whenever judged necessary to fac[illegible] diagnosis of deafness Blood groups of the parents were [illegible]

*tela olo* [illegible]

TABLE IV 2 *EEG Classification of 500 Deaf Children*

| | |
|---|---|
| 1 *Slow wave activity* | |
| A Group 1 Posterior slow wave | 15 |
| B Group 2 Diffuse slow wave activity | 31 |
| C Group 3 Paroxysmal slow wave activity | 24 |
| | 70 |
| 2 *Normal EEG and other pathologies* (Abnormalities) | 430 |
| Total | 500 |

The study concerns 500 EEG recordings to determine whether there is a pathological pattern related to deafness and retarded speech All the EEG tracings were first interpreted by one electroencephalographer and they were then reviewed and discussed with a second interpreter The clinical data were not made available to the second interpreter in order not to influence his classification

*EEG Classification* The classification is based on the distribution and morphological aspect of the slow wave abnormalities All EEG's presenting an abnormality other than slow wave activity were excluded from this study A review of 500 EEG s showed 70 recordings presenting slow wave activity

We further divided this slow wave activity into three groups according to their distribution (See Figs 10, 11 and 12 )

*a*) group I posterior slow wave activity

*b*) group II diffuse slow wave activity

c) group III paroxysmal slow wave activity (localized and diffuse)

In the first groups are included only those tracings in which the slow wave activity is limited to the posterior head regions and at no time is observed over other regions Another characteristic of this group is the blocking effect upon opening of the eyes, in other words this slow wave activity disappears upon opening the eyes During hyperventilation this slow wave activity is still limited to the posterior head region The etiological cause of deafness for this group is shown in Table IV 3

The second group of abnormalities, which we call diffuse slow wave activity, is not limited to the posterior head regions The EEG's show some generalized sharp and slow wave activity of high amplitude which at

TABLE IV 3 *Group 1 "Posterior Slow Wave*

| | |
|---|---|
| Genetic deafness | 2 |
| Unknown | 7 |
| Cerebral damage | 6 |
| Total | 15 |

on the part of the subject and they do not require any training or conditioning, but they do require either an experienced observer or, as we shall see later, a computer to extract the relevant information from a welter of irrelevant ongoing electric activity. To open our discussion of electroencephalography I will ask Dr Bertrand to say a few words concerning the broad relations between impairments of hearing and the types or classifications of EEG

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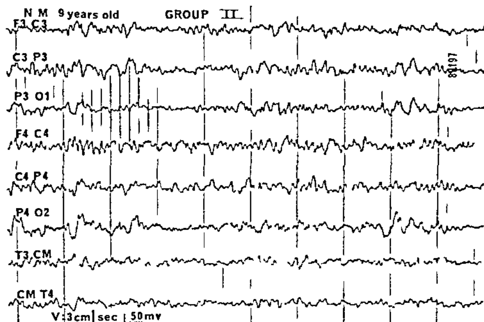



Fig IV 11 GROUP II In this group the slow wave is not limited to the posterior head regions is poorly organized and more or less influenced by hyperventilation

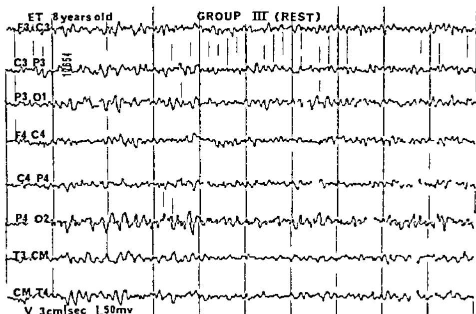



Fig IV 12 GROUP III E T., 8 years old Short paroxysmal bursts of slow wave activity are seen in all regions at rest This activity is increased by hyperventilation

in many children during the maturation of the brain the persistence of this anomaly with such regularity in our series where the average age is between 6 to 15 years of age must be taken into consideration

TABLE IV.5 *Group 3 "Paroxysmal Slow Wave Activity" (Localised and Diffuse)*

| | |
|---|---|
| Genetic deafness | 3 |
| Unknown | 5 |
| Cerebral damage | 16 |
| Total | 24 |

There exist differences of opinion between several authors concerning the frequency with which abnormalities in posterior head regions are encountered in normal children

Gibbs, Gillen and Gibbs (1954) and Blanc *et al* (1959) have presented evidence that occipital foci are not only the most common type of foci occurring in the infant and very young child but also that there is tendency for these foci to disappear in early childhood Gibbs *et al* in a series of 45 cases found that in only 23% was the occipital focus still present at the age of nine.

In a study of 915 children under the age of sixteen with eye disorders but no evidence of brain injury, Levinson and Stillerman (1950) showed 23% incidence of occipital electrographic abnormality as compared with only 0 6% in normal controls

In children with both ocular disorders and brain injury the incidence of occipital foci rose to 85%. Other papers in relation to occipital foci and eye disorders are reported by Kellaway, Bloxsom and MacGregor (1955), by Gibbs, Fois, Gibbs (1955), Jim and Kraus (1954), Parmelee, Fiske and Wright (1959), Cohen *et al* (1961), and Gastaut (1961)

The presence of these localized abnormalities in relation to specific end-organ disorders in the eye can lead us to wonder if a lesion of another end-organ, the ear, could not also show abnormalities in its projection on the cortex

In regard to the posterior localization of these waves, a presentation of 452 cases of occipital foci was presented by Smith and Kellaway (1963), who found the following clinical correlations

Ocular abnormalities—61 cases or 13 3%
Seizures—254 cases or 56 2%

Other signs and symptoms of cerebral disorders

Mental retardation—121 cases or 26 7%
Retar[illegible] speech—42 cases or 9 2%

It is th[illegible]p of retarded speech which attracts our attention

In our [illegible] cases of retarded speech, usually associated with hearing [illegible] EEG's presented slow wave activity in the posterior head regions [illegible]es even extending to other regions This is much

above the percentage of 0 6 found by Levinson and Stillerman in norm subjects

We believe that in some cases this slow wave activity is related to th clinical symptoms of retarded speech possibly as the result of the nor maturation of the cerebral cortex of a certain area due to the absence of th peripheral aural stimuli

As stated by Barnet, retardation of visual or auditory stimuli can resu in a permanent defect of these affected organs which can never develo to their full potentiality

In order to explain the persistence of the abnormal EEG we are face with several possibilities We believe that the three groups of slow wav activity could possibly be of different etiology

The first group of localized posterior slow wave activity seems to us t be either 1) from non-maturation of the cerebral cortex, 2) from a possibl biochemical error or metabolism, or 3) from localized cerebral damage

In the second group of 31 cases, the diffuse slow wave activity is no localized to the posterior head regions The etiology of cerebral damage i much more pronounced in this group and the fact that it is influenced b hyperventilation can lead us to believe that the pathology is sub-cortical

The third group of abnormalities consists of paroxysmal and occasionall rhythmic slow wave activity, more pronounced over the posterior hea regions at rest but recorded diffusely in the form of short bursts upo both hemispheres These anomalies are highly suggestive of subcortica damage Again the history of cerebral damage is more frequent than in th first group

*Conclusion*

1) We find a higher percentage of slow wave activity in deaf childre in the posterior head region than in normal children

2) Three different patterns of abnormality are found in relation t posterior slow wave activity

*a*) Posterior slow wave localized in the posterior regions disappearin on opening of the eyes, and not influenced by hyperventilation

*b*) Diffuse slow wave activity

*c*) Paroxysmal slow wave activity (localized and diffuse)

3) We believe that these posterior slow waves are pathological and car result either from cerebral damage or possibly from non-maturation o certain specific areas of the cerebral cortex due to the lack of a specifi stimulus, in this case the auditory stimulus, or finally from a possible non identified biochemical error

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CHAIRMAN Now I will ask Dr Derbyshire to tell us about evaluation of auditory function by direct observation of the EEG

## *Electroencephalic Audiometry (EEA)*

DERBYSHIRE In approaching the measurement of a sensory function there are two major problems to be solved One concerns the nature of the measurement itself and the other concerns the relation of the measurement to the sensory function I would like to discuss both of these questions as they relate to the evaluation of 'hearing' derived from direct inspection of EEG changes recorded during intermittent acoustic stimulation

### *Auditory Function Evaluated by Direct Observation of EEG Responses*

This report is based on work carried out at Harper Hospital in Detroit with Dr M McDermott Mr A Fraser and Miss A Bridge, at Parmly Hearing Institute at Loyola University Chicago with Dr J C. Farley, Dr P S Dhruvanajan Mr R Oppfelt and at the University of Illinois at the Medical Center Chicago with Mr C W Palmer A Lee and Dr C Elliott The latter part of this work was supported by NIH grants #B-2467 and NB-04134-01 and Grant 2-41 33 78 3 81 Graduate Research Fund of the University of Illinois Graduate College

### *Meth d*

The r u l single placement for electrodes for detecting changes of EEG in n ustic stimuli is one at a point 3 cm lateral to the

midline in the interaural plane and the other on the opposite mastoid or ear lobe The subject may be awake or asleep but preferably he will vary widely between sleep and wakefulness during the one and one-half hours of recording required for a useful study If a sedative is used, a moderate dose is better than a heavy one as the latter obscures the responses

For stimuli we use pure tones (of 0.5 to 4.0 sec duration the latter preferred with rise-decay times of 10 to 25 milliseconds vibrations, clicks and spoken words The acoustic stimuli are presented through earphones or in free field

An audiometer with adequate grounding makes an effective stimulus generator It allows measurement of intensities of tones and, through the speech circuit, the intensities of clicks and words. We use primarily intensities of 20, 50 and 80 dB (ISO) and by means of a special attenuator —50 dB, which is well below threshold. We concentrate our efforts on three pure tone frequencies, a low tone (250 cps), a mid tone 1000 cps and a high tone (4000 cps

To signal the stimulus we use an amplifier whose output is the integral of the input. The output of this amplifier is recorded on one of the EEG channels The input of this amplifier is a derivative of the electrical signal of the stimulus before attenuation. A microphone near the subject's ear may be employed as input to this amplifier when testing in free field. The stimuli are presented in random order to preserve their novelty and the intervals between stimuli are randomly varied between 10 and 20 seconds.

The parameters of each stimulus are written on the record with a red pencil When the record is read later through red goggles so-called "dark adaptation goggles" this information is invisible and does not bias the scoring

*Scoring the Responses*

The scientific essence of this method as used by us and by Rosenblut and associates 1959 is the unbiased scoring of the changes evoked in the EEG by supra- and sub-threshold stimuli. The pattern is inspected for any change a K-complex, an on-effect, an alpha block or an increase or decrease in amplitude of the total pattern or any component of it. The magnitude of the score depends on the amplitude of the change and the certainty of the reader that he has identified a change

These changes are looked for separately in each of three time zones. The degree of change is scored on a preset scale (Derbyshire and Farley 1959)

If the stimulus is less than 1.0 sec in duration only the onset response scale 1 through 5 can be evaluated.

The average scores for each intensity-frequency combination are determined and compared to the scores for all the —50 dB presentations.

*Scale for Scoring*

| | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| 1 Within $1^1/_2$ sec of onset of tone | no change | doubtful change | probable change | definite but not classifiable | obvious and classifiable change (K complex etc) |
| 2 During stimulation from $1^1/_2$ sec after onset until termination | no change | change | | | |
| 3 Within $1^1/_2$ sec after termination of stimulus | no change | possible change | definite change | | |

There is a consistent probability (around 20 per cent) that EEG changes will occur when a subthreshold stimulus (−50 dB) is presented These take place 1) because of the chance occurrence of an EEG change at that time 2) because of an EEG change due to some extraneous uncontrolled stimulus over any modality and 3) because of errors of judgment of the reader For these reasons a statistical test is necessary for reliable demonstration that a response exists

One can use Student s *t* test for this comparison Response is accepted as present if the scores are different at a 10 per cent level of significance (Walker and Lev) At this level of difference there are only 10 chances in 100 that the responses to the two different stimuli belong to the same population

It requires about two hours to obtain a complete study and about two to three hours to score and calculate results The expense however of equipment is only that of the EEG and the audiometer No special expert capacity of the observer is needed to score these records in this manner Self training is available by studying several normal subjects In such a study one learns what changes in pattern are best to rely on to differentiate a response from background activity The criteria do show some variation between readers but good correlations ($r$ 0 7) between observers is possible

Recently we have been able to define the change of pattern at the onset of stimulation which is specific to the state of repose (the evoked potential) This definition is operational enough to be taught to a new observer at one sitting The following procedure applies to EEG records that are made with the upward movement of the pen indicating greater electrical negativity of the vertex region relative to the mastoid region A ruler is laid on the EEG tracing parallel to the time axis and is moved downward from above unt one identifies the tallest negative peak that lies between 3 millimeters and 15 llimeters after the signal marks the onset of tone At a tape speed f 60 mi rs per second this corresponds to a latency between 50 and

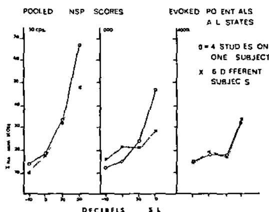



FIG IV 13 EEG responses to pure tones scored on scale of 1 5 for on effect (or evoked potential) as obtained by direct inspection of the record Mean score at each intensity presented as percent of maximal score possible

Rise time = 15 msec., Duration – 500 msec., Interval – random Alert repose and sleep states of subjects combined

250 milliseconds The ruler is then lowered still further until the lowest positive trough following the negative peak is identified in its turn This positive peak must occur within one half second after onset of stimulus If such a pair of maxima first negative and then positive, is found within the prescribed time intervals a response is accepted as present when the amplitude of the negative peak to positive trough is greater than the maximal amplitude in the immediate pre stimulus pattern The peak to trough amplitude can be converted to a score (1 through 5) This rule is applicable to subjects in repose between 4 and 60 years of age The report of Goodman, Appleby *et al* (1964) suggests that this same complex is pre sent in the same time relations in neonates (see Fig 14) (See also the template described by Derbyshire and McCandless (1964) )

From the results of reading and scoring these EEG changes we conclude that responses at *20 dB (ISO)* signify normal hearing Responses only at 50 dB (ISO) and above mean some hearing loss and responses only at or above 80 dB (ISO) mean a severe hearing loss

One can estimate threshold for each frequency when several intensity levels are studied by determining the sloped line best expressing the trend of the scores at intensities that are significantly different from sub threshold stimulation Where the extrapolation of this sloped line crosses the level of scores for sub threshold stimuli may be accepted as one estimate of threshold An alternate method of estimating thresholds is given by Rosen bl it *et al* (1959)

A linear trend of increasing scores with increasing intensity is not always ev ent except in studies with a large number of observations (see Fig 13 l also the report by Dr Rapin in this symposium) Therefore this thresh

*Scale for Scoring*

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|---|---|---|---|---|---|
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*One can use Student's t test for this comparison Response is accepted as present* if the scores are different at a 10 per cent level of significance (Walker and Lev) At this level of difference there are only 10 chances in 100 that the responses to the two different stimuli belong to the same population

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about their EEG changes by utilizing the same reticular mechanism from brain stem to cortical levels as the acoustic stimuli

The system we follow for interpretation of where an auditory loss occurs is based on the following criteria

1) Abnormality of basic EEG pattern signifies a cortical or corticothalamic disturbance (local or diffuse, constant or paroxysmal) Such a disturbance could interfere with cortical processing of auditory data

2) Lack of responses to sound and vibration in only one area signifies a local cortical or subcortical disturbance

3) Lack of all responses, vibratory and auditory, signifies a disturbance in the reticular system

4) The combination of absence of auditory responses and presence of normal vibratory responses signifies a peripheral disturbance in ear or auditory paths at the medullary level

Two questions remain unanswered The first is whether the absence of an EEG response to a sound implies that the subject is deaf to it in the sense that he cannot report hearing any such tone The physiologic basis for hearing might still exist in a non-detected portion of the cerebral electrical activity The converse is also a problem If a subject gives an EEG response, does this mean that he hears the sound in a way that is useful to him and conveys information to him? Normality of all the electrophysiological measures does not rule out disturbances in areas not recorded or detectable by our present means The subject might still have a hearing deficiency

It is true, on the average, that there is good agreement between standard audiometric threshold and EEG threshold depending upon subject (S D ±6 to 18 dB) In specific situations, however, there are many discrepancies of considerable interest For example, children who have had no auditory training tend to have lower EEG threshold than standard audiometric thresholds This difference disappears after training The standard threshold becomes lowered Another example occurs in normals at the time of falling asleep the standard audiometric threshold may rise 5 dB while at the same time the EEG threshold is improved by 5 dB

These discrepancies are ways in which EEG responses provide us with information If EEG and standard audiometry yield only the same results, then standard audiometry is much easier to perform and EEG offers us nothing more except perhaps a method for use with newborn infants or non-communicative subjects But since there are discrepancies, there is more information to be gained from both than from either standard audiometry or EEG study alone It is for this reason that we have never offered a statistical study of the correlations of EEG thresholds and audiometric thresholds They are, in our mind two different entities and cannot be readily compared A study of pure tone thresholds alone is comparable to evaluating a farmer's field by examining its fences But an EEG study offers us more than just the thresholds of the function, namely, the whole

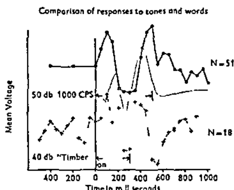



FIG IV 14 Comparison of Responses to Tones and Words
0—0 average of 51 responses in 4 normal subjects in states of repose to 1000 and 250 cps at 40 dB S L
average of responses of newborns to clicks The curve was copied from report of Goodman *et al* (1964)
+—+ average of 18 responses in one subject to the word 'timber" at 40 dB

range of auditory response with changing intensity These intensity functions cross the auditory field where hearing actually occurs in everyday communications The properties of this area are of prime importance and cannot be determined by the standard audiometric threshold

The EEG also gives us an opportunity to study the electrophysiology that accompanies conditioning (Gastaut, 1957) Dr Walter in this conference describes the CNV, a wave which relates to the development of expectancy and which can be conditioned In addition, we have been able to record the responses to words (Fig 14) These responses are similar in form to the responses to tones but their presence opens the possibility of studying the effects of semantic content of auditory stimuli In fact, EEG audiometry is like a sound probe by which we can begin an exploration of the dimensions of the cerebral caverns through the forms of their electrical echoes

CHAIRMAN Thank you, Dr Derbyshire, for your description of the various types of EEG response to sound and the way in which you recognize them I believe we shall hear shortly how the use of the average response computers makes it easier to detect one variety of such response, namely the cortical evoked response Your method of inspection of the original EEG record can detect other indications, notably the blocking of ongoing rhythmic activity, and may therefore yield different and additional information Thank you also for drawing the distinctions between impairments in the sense organs, in the intermediate auditory pathways, and in the responses of the cortex itself, and for reminding us that there is more than one pathway between the ear and the cortex These considerations ' qually pertinent for the interpretation of the results that several other s of the conference are about to report

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## *Average Evoked Responses (AER)*

CHAIRMAN A new possibility for electroencephalographic audiometry has been opened by the introduction of the technique of averaging the responses to many stimuli by means of a computer This is a logical extension of earlier efforts at graphic or photographic superposition of EEG traces but it proves to be far more effective Our next speaker Dr Rapin will tell us something about both the technique and also the phenomena as seen in normal adults

## *Auditory Evoked Response in Normal Waking Adults*

A Parametric Study of the Auditory Evoked Response in Normal Waking Adults—A Preliminary Report I Rapin I M Tourk N A Krasnegor, and H Schimmel of the Saul R Korey Department of Neurology, Albert Einstein College of Medicine New York New York This work was supported by grants NB 2503 NB-3356 and 2T1 NB 5325 from the National Institute of Neurological Diseases and Blindness United States Public Health Service

RAPIN The evoked response to auditory stimuli which can be recorded on the scalp holds considerable promise as an objective test of hearing in selected children suspected of hearing losses in whom a diagnosis cannot be firmly established by conventional audiometric methods (Rapin, 1964) Previous papers have described some of its characteristics in normal waking and sleeping subjects, both adults (Williams *et al*, 1964, Weitzman and Kremen) and infants (Weitzman *et al*) It was necessary to study systematically its characteristics as a function of stimulus parameters, its stability in a given subject, and its reproducibility from subject to subject before starting to use it routinely for diagnosis

*Method* Subjects were three cooperative adults whose hearing was tested by pure tone audiometry They were seated in a sound treated room and were instructed to listen attentively to the stimuli without counting them or making any overt or covert motor responses

All stimuli were presented monaurally to the left ear They were clicks produced by a 0 4 msec square wave presented to the amplifier, attenuator and earphone, and tone bursts at 250 cps, 1000 cps, and 6000 cps The envelope of the tone bursts was a trapezoid whose horizontal portion lasted 25 msec and whose symmetrical rise and decay lasted 15 msec for tones at 250 cps, and 10 msec for tones at 1000 and 6000 cps The subjective threshold for each of the stimuli was established on each experimental day using the method of limits Each of the stimuli was presented 100 times at threshold, 5 dB, 10 dB, 20 dB, 35 dB, and 50 dB sensation levels, and runs without a stimulus were also recorded These seven runs obtained in random order constituted a stimulus block Six blocks for each of the four stimuli were obtained The interval between stimuli was irregular with an average of 3 0 seconds

Scalp electrodes were placed one cm lateral to the vertex and were referred to the ipsilateral mastoid process The amplified EEG was sampled for 1000 msec following each stimulus presentation and summed by the Mnemotron CAT The average (sum) of 100 samples was written out by an XY plotter

The most constant components of the evoked response were a vertex negative wave ($N_1$) with a peak latency of 90 to 115 msec and a vertex positive wave ($P_2$) with a peak latency of 170 to 200 msec The latency and the amplitude of the two peaks were measured and analyzed

*Results* There was no difference in the latency or the amplitude of the activity recorded from the two sides of the head The morphology of the response to clicks and to tones was similar, and showed relatively minor variation among the subjects Evoked potentials were recognized and measured in all subjects down to 5 dB sensation level for each of the stimuli but not for each presentation of that stimulus intensity The amplitude of the response decreased with stimulus intensity falling from about 8 20 microvolts at 50 dB sensation level down to about 3 10 microvolts at 5 dB sensation level (Fig 15) At low stimulus intensity there was some un

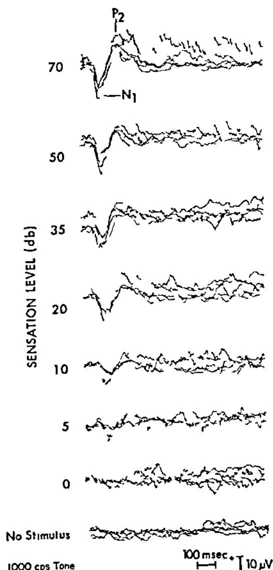



Fig. IX 15 Evoked responses to 1000 cps trapezoid tone bursts at seven intensity levels in one subject Each trace represents the sum of 100 epochs lasting 1000 msec each Upward deflection represents positivity at the vertex

certainty in distinguishing the evoked response from the ongoing background activity particularly when the latter was high In such cases measurements were not made

The most striking finding pertained to the latency of both $N_1$ and $P_2$. It was remarkably constant for clicks throughout the range of intensity levels The latency of the response to tone bursts was 10 msec longer than the latency for clicks at 50 dB sensation level and increased progressively

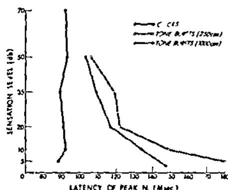



Fig IV 16 Mean latency of peak $N_1$ as a function of intensity in three subjects

as the tones were attenuated being 25 to 60 msec longer at 5 dB than at 50 dB sensation level (Fig 16)

*Discussion* The response obtained is similar to that recorded by other investigators in the waking state and in sleep It occurs later than the myoclonic response described by Bickford (1964) The lack of difference between the sides of the head ipsilateral and controlateral to the stimulated ear is not unexpected with a response originating at the vertex This electrode placement might miss minor asymmetries occurring in the vicinity of the primary auditory cortex, but we have not recorded a primary auditory evoked response nor have we found in the literature a description of one recorded from the human scalp

The most easily recognized and reproducible elements of the response over the range of stimulus types, intensities, and subjects were the waves $N_1$ and $P_2$, using the nomenclature of Williams (1964), of Weitzman (1965), and of Davis (1963), and their collaborators Wave $N_1$ is the most easily measured element of the response because it usually comes to a sharp peak $P_2$ may be as large or larger in amplitude but because it is more rounded it tends to lose its measurability with low level stimuli Earlier and later components can often be recognized but they are less constant

Psychophysical experiments have shown earlier that temporal integration plays an important role in auditory perception In such experiments a critical duration curve was found above which subjective loudness is independent of the duration of the sound and below which sounds with equal sound pressure are heard as louder if they last longer (Miller, 1948) The critical duration is longer for sounds near threshold than for louder ones, indicating that temporal integration increases in importance as threshold is approached The increasing latency of the evoked responses as intensity decreases is the electrophysiological reflection of this phenomenon in man, we think When very short clicks are used, there is virtually no opportunity for temporal integration, and the latency of the evoked response remains constant With the trapezoid tone bursts, temporal integration occurs during the rise time of tones at all intensities, as shown by the slightly greater

latency of the evoked response at 250 cps where the rise time was 15 msec contrasted to the latency at 1000 cps where the rise time was 10 msec It is shown also by the longer latency of $N_1$ for tones than for clicks (Fig 16)

The auditory evoked response is thought to be an on response (Goldstein & Kiang, 1958) This idea is supported by our data showing that whether clicks or tones are used its configuration is stable and remains so even when its amplitude decreases and its latency shifts

*Summary* The auditory evoked response recorded near the vertex is reproducible in a given subject and varies relatively little among normal subjects making it a suitable and reliable indicator of the functioning of the auditory pathway Its configuration is stable whether clicks or tones are used and whether it is recorded ipsilaterally or contralaterally to the stimulated ear Although its amplitude becomes small it can be seen quite regularly with stimuli at *10* dB sensation level and not infrequently at 5 dB sensation level As the intensity of clicks is decreased its latency remains constant whereas with tones the latency increases as a function of decreasing intensity This observation can be related to the occurrence of temporal integration which increases in importance as intensity approaches threshold

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## *Practical Considerations in Using the Evoked Potential Technique for Audiometry*

This work was supported by grants NB 2503 NB 3356 and 2T1 NB 5325 from the National Institute of Neurological Diseases and Blindness United States Public Health Service

Rapin Clearly discernible auditory evoked responses can regularly be recorded from the scalp of cooperative waking adults in response to clicks and tone bursts 20 dB above subjective threshold whereas they cannot al

ways be picked out from background activity with stimuli at 5 and 10 dB sensation level (Rapin, previous presentation). More complex responses with higher amplitudes have been recorded during sleep in infants and adults (Weitzman *et al*, Williams *et al*, 1964) but a systematic survey of the effects of stimulus attenuation upon parameters of the response during sleep has not yet appeared The recording of a response on the scalp is a clear indication that the ear and auditory nerve have been stimulated and that the stimulus has reached the central nervous system, it does not necessarily indicate that the primary auditory pathway or auditory cortex are functioning nor that the stimulus has been "heard", that is that the subject perceived it and can utilize it to modify his behavior. This limitation does not detract from the unquestionable value of the evoked potential method for audiometry in selected patients, notably in young children in whom the urgent concern is to recognize a peripheral hearing loss early so that suitable amplification and auditory training can be instituted without delay.

Our experiences with a range of patients, most of whom had no previous experience with auditory testing, have highlighted some of the practical problems to be expected when using this method in an ordinary clinical setting We felt that it might be of some value to discuss them and to formulate a few suggestions directed at achieving optimal efficiency of the method for audiometric purposes

*Subjects* We have reported elsewhere our attempt to establish threshold in 50 children attending St Joseph's School for the Deaf (Rapin, 1964) Subsequently we have studied eight normal adults and 41 patients from the wards of the Bronx Municipal Hospital Center, eight of these were infants, 28 were children aged 4 to 14 years, and five were adults Two adults, two infants, and four children were known to have cerebral pathology

It was not possible to obtain formal audiograms in most of our subjects but they were not suspected of having hearing losses We attempted to determine subjective auditory threshold at the time of testing and we referred stimulus intensity to this threshold In uncooperative subjects, in children who gave obviously inadequate threshold values and in infants stimulus intensity was referred to subjective threshold of adults with normal hearing Most subjects were tested in the sitting position An observer usually sat with the child in the sound treated room encouraging him to read or to look at pictures, and coaching him not to move during actual recording A few subjects were tested recumbent, all of the infants were lying in their cribs

*Stimuli* Clicks were produced by presenting an 0 4 msec square wave to the earphones or to a speaker Tone bursts were produced by an electronic switch which shaped the envelope of the sine wave from an audio oscillator into a burst with a symmetrical rise and decay lasting 10 msec for frequencies above 1000 cps and 15 msec for lower frequencies, and a constant portion lasting 25 msec We wished to obtain a tonal stimulus of

short duration without "clicky" characteristics, that is with as few transients at other frequencies as possible Intensity of the stimulus was controlled by using an attenuator in series with an amplifier

In waking subjects the stimuli were delivered by earphones This permits monaural stimulation and rather precise control of the stimulus In sleeping subjects and in infants the stimuli were delivered by a loudspeaker Calibration was then carried out by placing the experimenter's head at the place of the subject's head and determining his threshold Changes in position of the subject's head with respect to the speaker no doubt result in changes in loudness, but it is unlikely that their magnitude invalidates the use of a loudspeaker to estimate threshold in infants and during sleep if a precision no greater than 20 dB is required

*Response* The response was obtained from surface electrodes at or near the vertex referred to the mastoid process A one second sampling time was used and averaging was done using the Mnemotron CAT In waking normal adults and children, the most useful portion of the response was a vertex negative wave occurring about 100 msec following the stimulus and a vertex positive wave occurring at about 200 msec The amplitude between these peaks fell from 20–30 microvolts at 50 dB sensation level down to 8–15 microvolts at 20 dB sensation level when 100 stimuli were used Amplitude was greatest for 1000 cps tones During sleep, a vertex negative wave at about 400 msec and a vertex positive wave between 600 and 900 msec were most prominent

*Rate of Stimulation* Slow rates of stimulation and irregular spacing of stimuli were found by us and by others to yield responses of higher amplitude for a given number of stimuli than faster regular rates, a very important consideration when working near threshold We used a regular rate of 1 per 3 seconds, or more often an irregular rate averaging 1 per 3 seconds produced by triggering the electronic switch or click generator by irregularly spaced pulses on magnetic tape This slow rate limited the number of traces which could be obtained at one sitting Recording the response to 100 stimuli took five minutes, in addition to which several more minutes were used writing it out and resetting the apparatus This enabled us to obtain 6–8 traces in one hour in a cooperative subject A more serious disadvantage was the tedium of the procedure Five consecutive minutes of comparative immobility appeared above the capacity of many children even at age ten years or above and of some adults who were not highly motivated to cooperate There is an optimum irregular rate, which we have not determined, which will minimize testing time without unduly sacrificing amplitude It is probably faster than one per 3 seconds and slower than one per second

*Number of Stimuli* We chose to sum the responses to 100 stimuli in every case With loud stimuli an evoked response is often clearly discernible after 30–50 presentations Using the smallest number of stimuli which yields a clear response is one obvious way of shortening recording time,

on-line monitoring of the response on the face of the computer's oscilloscope makes this quite feasible The technique of averaging is dependent on the fact that the amplitude of time-locked activity will grow in proportion to the number of samples ($N$) whereas random and rhythmic background activity which is not time-locked grows approximately as $\sqrt{N}$ The amplitude of the auditory evoked response is small with respect to background activity, particularly with stimuli close to threshold, since amplitude decreases with decreasing stimulus intensity Consequently a larger number of presentations is needed for the evoked response to emerge when the stimuli are weak than when they are loud

*Background Activity* Decreasing the amplitude of background activity by minimizing EMG activity is another way to enhance the evoked response and decrease the required number of stimuli Having the subject lie rather than sit accomplishes this and results in a clearer evoked potential High background activity was the single most frequent cause for the failure of a stimulus at 20 dB sensation level to elicit a recognizable evoked response

*Sleep* A possible solution to the problems of boredom, of movement and of muscle artifact is to record during sleep This method is eminently suitable for testing infants and young children who readily fall asleep during the day We obtained some clear responses at 20 dB sensation level in some children but do not have enough data to comment about thresold determination in infants Further studies concerning the effects of hypnotic agents and of varying stimulus intensity upon the response in sleep will have to be carried out in the various age groups

Our data show the advantage of recording in sleep, at least with children In waking subjects an average of 10 traces, with a range of 5–15, was obtained For the children who dozed off, the average number of traces was 13, with a range of 8–24 and for the infants, all of whom slept a substantial part of the time, the average number was 17 with a range of 9–23 Sessions with waking subjects lasted one and a half to three hours, including threshold determination and application of the electrodes When subjects fell asleep there was both a decrease in the time wasted coaxing the child to cooperate and an increase in the total time spent in the laboratory

There is a disadvantage to recording in sleep, however Williams (1964), Weitzman (1965), and others have shown that the latency, amplitude, and configuration of the auditory evoked response vary considerably during the various phases of sleep The amplitude of the auditory evoked response decreases markedly during the rapid eye movement or activated phase of sleep which comprises 50% of sleeping time in infants and 20% in adults Clinicians planning to record during sleep should familiarize themselves with these variations during sleep It may be necessary to follow the recommendations of the above mentioned investigators who continuously monitor with conventional EEG during sleep so that the phase during which a given evoked response was recorded can be determined

*Summary of Results* Satisfactory recordings were obtained in seven of eight infants and in all the normal adults The data obtained in six of 32 patients had to be discarded because background activity was too high or because of excessive movement artifact Among the records of the 26 remaining patients, some single traces were uninterpretable for the same reasons One nine year old child with degenerative brain disease and grossly disturbed behavior was untestable on two occasions despite the use of sedatives

In contrast to the children without previous experience of auditory testing, 48 of 50 children from St Joseph's School for the Deaf were able to cooperate to the extent that some satisfactory tracings were recorded from each of them In 37 of the 50 enough data were obtained to make some statement about threshold Three additional children from the school aged seven years, were untestable without sedation which was not used

We conclude that when recording circumstances are favorable, responses to clicks and tones at 20 dB sensation level are clearly seen in normal children as well as in normal adults in the waking state, and in at least some phases of sleep The same seems to be true in patients with hearing deficits using clicks, we cannot say whether this is true of tones also since we have not used them in such patients

*Suggestions for Testing Clinical Subjects* Recording in the recumbent position is desirable because it minimizes EMG activity and because it favors spontaneous sleep Inducing sleep is the obvious solution in dealing with infants, young children and incooperative subjects Sleep has the added advantage of prolonging the amount of time during which the subject is available for testing It is probably wise to monitor the EEG during sleep because of variations in the shape and amplitude of the response, notably during the rapid eye movement phase of sleep which occupies a large portion of sleeping time in infants and young children

Efficient use of time is the key for making this method clinically useful Stimuli should be presented at irregular intervals The most favorable rate remains to be determined, it is probably longer than 1 per second and shorter than 3 per second The building up of the response on the scope should be monitored so that the smallest number of stimuli yielding a clear response can be used An unequal number of stimuli in different samples is not critical since amplitude is useful only in a general way, to indicate whether the stimulus is close to or far from threshold

It is important to select carefully the levels of intensity with which to start testing There is only time for presenting a given stimulus at two, or rarely three, levels to each ear if useful screening at three frequencies is to be accomplished in one sitting Having to present an intensity level twice because the response obtained was equivocal is so costly time-wise that attempts at a precision greater than 20 dB are unwarranted

If it is possible to get some idea of subjective threshold, one might start 20 dB above that level, increasing or lowering intensity for subsequent

traces depending on whether or not an evoked response was obtained In children suspected of profound losses, it would seem reasonable to start with the loudest stimulus available When no information is available, one might start at 50 dB, increasing to 80 dB or decreasing to 30 dB depending on whether or not a response was seen It is generally inadvisable to present the same stimulus repeatedly at decreasing intensities, randomizing both frequency and intensity seems to minimize the number of false negatives, that is tracings with no apparent response at an intensity level which has already yielded a response or does so later

The choice of frequencies will depend on the anticipated type of hearing loss, keeping in mind that three frequencies at two levels for each of the ears may be all that can be obtained Since the amplitude is higher for 1000 cps tones than for 250 cps and 6000 cps and for clicks, it would appear logical to start with that frequency

The aim of the entire procedure is to obtain two or three *valid* points on an audiometric curve, accepting an uncertainty of 20 dB around each point If this can be done, a significant step forward in assessing the function of the peripheral auditory apparatus of non-verbal children will have been taken

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CHAIRMAN *Thank you, Dr Rapin Now to explain the new term "sono* motor reflex" and to explain further the concept of "average electrical evoked *responses to sound , as revealed by computer techniques, I will call* upon myself as an investigator to summarise some of the background information

## *Sonomotor Reflexes Myogenic Evoked Potentials*

DAVIS The computer technique of summing a large number of electrical responses to clicks flashes of light, electric shocks, or in fact any stimulus that is clearly defined in time, goes back to Dawson (1950, 1954) Before this we had only the cruder and more limited techniques of simply superimposing EEG tracings, either ink-written or photographic It is not practical to superimpose more than 10 or perhaps 20 responses, even photographically [illegible] lectrical methods now allow us to accumulate as many

as we wish, whether it be a dozen or a thousand or more The essential point is that the response, if it is time locked to the stimulus, gradually accumulates and grows larger while the background "noise" that is not related in time to the stimulus and is more or less random in character tends to cancel itself out and approach zero It does not actually reach zero but the signal to noise ratio is greatly improved and it is quite practical to recognize responses, derived from scalp electrodes, that are only 5 to 10 microvolts in amplitude, even in the midst of a spontaneous electro-encephalogram with waves up to 100 microvolts or more

When this technique became available the first effort was to detect evoked responses in the primary projection areas of the visual, cutaneous and the auditory systems The attempt was successful with the visual and the cutaneous systems Waves with latencies of the right order of magnitude, namely from 20 to 50 milliseconds, were found in the appropriate areas of the scalp over the primary projection areas for vision and for touch The results for the auditory system were more equivocal, however Small responses were indeed detected at least in some subjects and under certain conditions but they seemed to appear most strongly from the wrong areas namely low in the occiput or from just behind the ear The auditory stimuli that evoked these fast responses were usually clicks, repeated at frequencies of 5 to 10 per second, and often the responses to several hundred clicks were averaged to obtain a clear pattern

Finally Bickford and his collaborators demonstrated quite conclusively that most, if not all, of these early responses to acoustic stimuli were actually myogenic in origin They appeared most clearly from the muscles attaching to the base of the skull at the back of the neck but might be detected in other muscles, even in the arms and legs They may appear in the jaw muscles as well They are enhanced by any maneuver that increases the resting tone of the muscles from which they originate In fact this dependence on muscular tone is one of the strong arguments for their myogenic character Bickford coined the term "sonomotor" to designate this reflex response, which is quite reproducible and which is characterized by a very short latency It apparently represents some quite simple and fundamental reflex motor pattern, probably related to orientation but perhaps representing merely a facilitation of ongoing tonic activity With strong clicks the response may evidently be mediated through the non-auditory part of the labyrinth, although it is our own conviction that at more moderate levels of stimulation the cochlea is the normal pathway

Quite recently Jacobson (1964) has shown that the source of responses that appear from the region just behind the ear is actually the post-auricular muscles This acoustic-auricular reflex in the guinea pig has been known for years as Preyer's reflex and, like the tympanic reflex, it has a particularly short latency of about 5 or 6 msec (Totsuka, Nakamura and Kirikae, 1954)

In the fall of 1962, when Dr. Yoshie, Dr Mast, and myself were busy

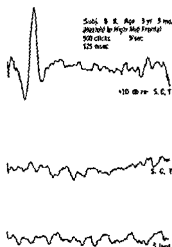



Fig IV 17

investigating these responses and confirming the myogenic interpretation of Bickford, Dr Lowell of the John Tracy Clinic joined us for a time He already had some experience with the computer method (Lowell *et al*, *1960*), *and following his return to Los Angeles he continued to investigate* what we call the "fast" or myogenic auditory evoked response

I wish to emphasize the theoretical importance of this response It involves only the ear, the brainstem and certain muscles of the ear, the jaws or the neck. It does not involve the upper auditory pathways or the cortex its latency is far too short for that Therefore, in principle at least, it may serve to differentiate failure of response due to impairment of the higher levels of the brainstem from impairments of the cochlea In this context it becomes the "opposite number", so to speak, of the cortical *response evoked by vibratory or by tactile stimuli mentioned by Dr Derby*shire. At this point I yield to Dr Lowell, who will tell us more of his experience with the "fast" auditory evoked response and whether he finds it useful as a method of audiometry for young children

Lowell I will say at the outset that I am not at all convinced that this fast response that I will describe is the most suitable for measuring the hearing of very young children. Furthermore, I am not entirely satisfied with our Chairman's description of this as a purely myogenic response I do agree, however, that the response does behave in a fashion that is consistent with its interpretation as an electroencephalographic representation of the orienting response. (I use the term "orienting response" in the Russian sense of alerting, rather than in the sense of turning the head toward a source of sound as in the Ewing tests of hearing )

We apply our *electrodes to the mastoid process and to a mid-frontal* reference point We deliver clicks at the rate of 5 per second and average the electrical output over a period of 120 milliseconds following each click

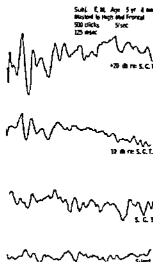



Fig IV 18

We usually sum the responses to 500 clicks We find a relatively stable polyphasic response with a mastoid negative peak of about 16 milliseconds and a second negative peak at 36 milliseconds and vertex-positive peaks at about 24 and 45 milliseconds The variability of these latencies increases with time following stimulation

Figure 17 shows the response of a child of 3 years and 3 months The response is clear at a level 10 decibels above the subjective click threshold In some subjects these responses are large enough to be clearly visible on the ink-written EEG record although this is not usually the case With this child we see nothing at threshold, and a control run at the bottom of the figure gives us the baseline for estimating the noise level

Figure 18 is the record from a child of five years and four months The response is clearly visible at about 10 dB above the subjective threshold There is little or nothing at the subjective threshold

Figure 19 is a little unusual because there is such a large response at threshold I suspect that the child had a little more hearing than our subjective click threshold revealed

In general the amplitude of these responses increases with the intensity of the stimulus We have tested very young children, even infants, successfully but we have come to the conclusion that the slow responses seem to serve us much better than the fast responses with very young children They seem to be more consistent

While I am on my feet I would like to show two samples of the slow response Our tracings are now 1000 milliseconds in duration The stimuli are given one every two seconds and we sum only 50 or perhaps 100 responses (Figures 20 and 21)

Figure 20 from an 11-month-old child, shows a fairly clear response

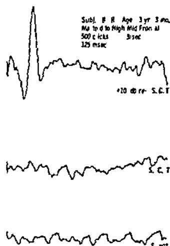



Fig IV 17

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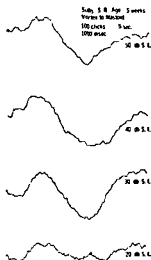



FIG IV 21

or children who are deaf from other causes, but the rather high variability of response latency makes it difficult to demonstrate statistically significant changes

GOLDSTEIN (in absentia) I, too, am not convinced that the faster early activity reported by Dr Lowell and seen by us under similar conditions is purely myogenic These are our conditions for recording the early components of the evoked response Bipolar recordings are made with the vertex referred to the earlobe, the forehead is grounded Two thousand click stimuli are presented at 10/sec The first 62 5 msec after the onset of the stimulus is analyzed

Under these conditions we often see a small response with a peak at 16–18 msec The vertex is negative with respect to the earlobe This is followed by a vertex-positive response with a peak at about 30 msec Several less consistent responses follow Even when the early negative wave is not seen, the beginning of the positive wave can usually be seen at 20–22 msec

Most of our observations have been made with a click at 50 dB sensation level Nevertheless, we have seen these early responses at lower sensation levels With me as a subject a small but clear positive wave was recorded at my voluntary threshold The foot of the response was at about 25 msec and the peak at about 34 msec

An electrode over the temporal region referred to the earlobe on the same side does not pick up the early negative response but does show the positive peak at approximately the same time as it is seen in the vertex-earlobe lead An implication of this observation could be that the vertex and earlobe electrodes may be looking at nearly opposite sides of the generator of the primary auditory response on the buried surface of the temporal

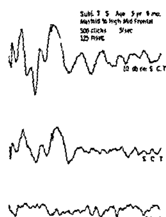



Fig IV 19

at 20 and even at 10 dB sensation level, and in Figure 21, which shows the responses of a five-week-old infant, there is a suggestion of a response as low as 20 dB I think that, as we gain more experience with this slow response recorded from vertex to mastoid, we should get results comparable to those of other investigators In the figures I have shown I would emphasize the apparent decrease in latency of the waves with age We find, however, that this change only takes place during the first year After that the latencies appear stable

There appears to be a tendency for children with clinically abnormal EEG's or other neurological impairments to give quite deviant responses In a group of 40 Rh children who were transfused at birth there appeared to be a somewhat shorter latency than in either normal-hearing children

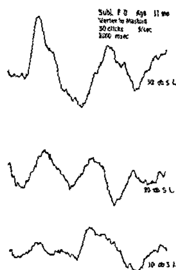



Fig IV 20

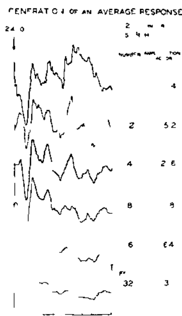


FIG IV 22 A series of 32 stimuli was interrupted to write out the cumulated sums as indicated The amplification was adjusted so that the calibration is the same for all of the records Note the progressive elimination of the spurious waves in the latter part of the early records The shrinkage of the average evoked response in the last three lines is due largely to the less frequent pauses for write out The response after a two second interval averages less than half the voltage of the first response after a long pause In addition this subject regularly shows considerable overall habituation during an experiment Upward deflection indicates vertex more positive relative to earlobe

animals that are used by electrophysiologists and neurosurgeons to map the sensory projection areas It does arise diffusely from the cerebral cortex over a wide area that is like a cap worn well forward on the top of the head Visual and tactile stimuli evoke very similar responses from the same non specific areas although there are minor differences from one modality to another and also from one subject to another and perhaps also with age in very young children

The most typical sequence of waves in response to auditory stimuli is a small vertex positive wave with its peak at about 50 milliseconds followed by a strong vertex negative wave at 100 milliseconds and then a large vertex positive wave at 175 milliseconds The final positive wave may be a plateau or show two peaks as in Figure 22 The latter part of the response may suggest a rhythm but it is much more variable than the initial positive negative–positive complex All of our quantitative measurements represent the peak to peak voltage from the first negative to the second positive wave We believe that the overall pattern is composed of three or four components that may vary independently in amplitude but which are fairly stable in latency It is this feature of being time locked to the stimulus that makes it possible to separate the evoked responses from the random

EEG background by averaging the electrical output after successive stimuli We also reduce the noise in the final records significantly by restricting the pass band for the initial electroencephalogram to the range from 0 3 to 35 c/s (half amplitude at these frequencies) The successive improvement in signal-to-noise ratio obtained by increasing the number of responses that are averaged is illustrated in Figure 22

The rate of stimulation must be no faster than one every ten seconds to obtain maximal responses The responses are very small and differ in waveform if the interval is only half a second This refractory effect operates within each sensory modality, i e sound depresses the response to a following sound and touch depresses the response to touch, even when delivered to opposite ears or hands, but touch, audition and vision interact very little with one another

In general, strong stimuli elicit larger responses but it is difficult to define a precise input-output relation because other factors, notably the state of the subject, whether vigilant or indifferent or habituated or drowsy, is important and also because of the variability of the responses, both across subjects and across successive trials Typical peak-to peak voltages in response to fairly loud clicks are of the order of 10 to 20 $\mu$v In rare individuals they may be as large as 100 $\mu$v and can be measured individually Successive responses vary by a factor of two or three and apparently quite at random, as shown in Figure 23

The long time-course and the slow recovery of the slow cortical evoked response, its variability, its complex structure, its curious anatomical distribution, and its semi-specific relations across sensory modalities make it impossible as yet to incorporate this response into any plausible scheme of the processing of auditory information It nevertheless can yield audiometric and perhaps neurological information of real clinical value, but only on a purely empirical basis

*Audiometric Use of Evoked Responses*

We have employed the slow cortical evoked responses as an end point in "objective" audiometry of severely hard of hearing children at Central Institute for the Deaf Our stimuli are filtered clicks or "tone pips", at five frequencies 300, 600, 1200, 2400, and 4800 c/s The pips all reach their maximum amplitude on the third wave and fall off almost as rapidly Thresholds were determined for normal listeners and compared with their audiograms This "threshold transfer" allows us to express our attenuator readings as hearing levels, i e decibels above the thresholds of an ideal listener whose threshold corresponds to the new International (ISO) reference zero level at each frequency

The children sit comfortably reading or are entertained with pictures Silver disc electrodes are attached at the vertex and either an ear lobe or the mastoid process Earphones (Philips) with circumaural cushions, rather than a loudspeaker, are regularly employed (except for children below

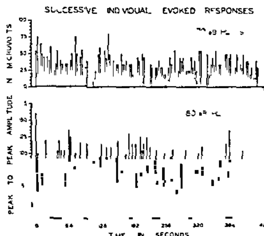



Fig IV 23 Unusually large auditory evoked responses could be measured individually in the standard EEG record of one normal subject The noteworthy feature is the large random variability of amplitude from one response to the next No cyclic or overall trends are evident The brief interruptions in the 70 dB series had no consistent effect Hearing Levels are relative to the ISO reference level The tone pips were 1200 c/s (From Davis, H., and Zerlin S "The Variability of Evoked Responses", *The Physiologist*, in press )

seven years of age) in order to reduce extra aural tactile stimulation Tone pips were delivered every 3 2 seconds and responses summed in blocks of 32 (We now believe that it will be more efficient to employ more frequent stimuli, perhaps at one per second, and to collect blocks of 64 responses ) The clarity and the amplitude of the response patterns are noted on the oscilloscope and graphic writeout, and the threshold for each frequency is estimated on the spot by extrapolation or interpolation Averaged responses at three well-chosen intensities are usually sufficient for a given frequency Since individual differences in pattern may be considerable, a clear supra-threshold response is required at the beginning of a series The first trial is therefore regularly made at 1200 c/s and at a hearing level of about 100 dB (ISO) Maximum hearing level available is 126 dB The entire procedure, including application of the electrodes for estimating a binaural audiogram for the five frequencies requires about 50 minutes

We have tested 60 pupils, most of them between 10 and 16 years old, 45 from the Oral Department and 23 from the Speech Pathology Department All of them are educationally deaf, but the pupils in the Speech Pathology Department have special difficulties in learning in addition to their hearing losses None of our pupils have pure "congenital sensory aphasia" A few tests were discarded as technically unsatisfactory Fifty of the children tested had sufficient hearing to give clinical audiometric end points, at least at 250, 500, and 1000 c/s In only one case did we fail to obtain electrical evoked responses at the two lowest frequencies, but we are ot yet certain whether the responses to the loudest sounds were truly

auditory or perhaps only tactile The child who gave no responses whatever has congenital atresia of the external ear canals All of the children who by clinical audiometry failed to hear 1000 c/s or higher were identified by the evoked-response test, without consulting the clinical record, as showing "probably only tactile responses (or none at all)".

After quantitative estimates of the evoked-response thresholds had been recorded, the voluntary thresholds for the same stimuli were then obtained The evoked-response thresholds were also compared with the most recent clinical pure-tone audiometric thresholds on record for the child, disregarding the small differences in the test frequencies employed

The distribution histogram of the (algebraic) differences between the evoked response and the voluntary subjective thresholds was normal in form (See Fig 24 ) The extremely deaf children are omitted in these quantitative comparisons The mean difference for the fifty with best hearing was −2 3 dB, i e the child's voluntary response was slightly more sensitive than our detection of the cortical evoked response This agreement is much better than for a control group of young adults for whom the mean difference is between −20 and −25 dB The scatter in the individual differences is somewhat wider for the children suffering from "speech pathology" than for those suffering simple, although severe, hearing loss

For the fifty children the difference between the evoked response thresholds and the clinical thresholds averaged less than 0 1 dB This exact agreement is clearly a coincidence as the distribution of differences is bimodal with peaks at approximately −9 dB and +5 dB (See Fig 25 ) Furthermore the arithmetical average of the errors of the individual estimates, frequency by frequency, was nearly 10 dB In two cases our average estimate for a child, at all five frequencies, was 18 dB or more in error (see Fig 25), but we nevertheless believe that the method has great potentialities for the assessment of the hearing of very young deaf children

*Summary*

Evoked responses of the waking human brain to acoustic stimuli can easily be recorded from external electrodes by means of an average response computer The clearest responses are anatomically diffuse (strongest from frontal and vertex), long in latency, and are evoked by visual or tactile as well as auditory stimuli Their amplitude varies quite widely across subjects, with the state of the subject, with the interval between stimuli (up to 10 seconds), and from one individual stimulus to the next The average of a set of successive responses is sufficiently related to the intensity of the stimulus however, to make the method useful for "objective" audiometry on a purely empirical basis, even though the response does not arise from the primary auditory cortical area

Our thresholds of detection of the average evoked responses of fifty severely hard of hearing children, 7 to 16 years old, were compared with the children's subjective thresholds for the same filtered clicks The mean

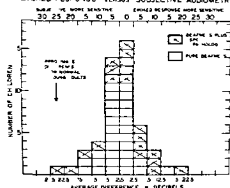



Fig IV 24

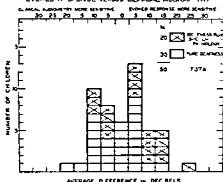



Fig IV 25

Fig IV 24 All of the 50 children are educationally deaf but all had clinical audiometric thresholds for at least 250 500 and 1000 c/s The differences represented here are between the estimated threshold of detection of an evoked response and the subjective threshold of the child listening to the same tone pips There were no false positive responses in the sense of an evoked response appearing at a frequency for which the child failed to respond voluntarily at all even though some subjective thresholds were less sensitive than the estimated ER thresholds The differences measured for each child were averaged algebraically The histogram shows the distribution of these average differences

Fig IV 25 This figure represents the same group of 50 children and evoked response tests as Fig IV 24 The difference in distribution is due chiefly to the differences in the acoustic stimuli tone pips (filtered clicks) versus pure tones Tone pips scatter acoustic energy and are therefore better heard than pure tones by children who have very steep audiograms either falling or rising Also the test frequencies differ slightly The clinical pure tone tests had been done as routine several weeks previously but the results were unknown to the electroencephalographers until after their estimates of evoked response thresholds had been recorded

difference was only 2 5 dB The averages for five frequencies diverged by 18 dB or more in only two cases The relation to their pure tone thresholds, as determined in our clinic, was almost equally close

## *Acknowledgments*

In 1958 Dr Atze Spoor explored, in our laboratory, several parameters of the evoked response( unpublished) He used a method of photographic superposition to obtain average responses Our present digital data processing computer was designed and constructed by A Maynard Engebretson and Donald Glaeser under the direction of Dr Jerome R Cox, Jr Mrs Shirley K Hirsh and Mrs Joyce Shelnutt assisted in obtaining the ER audiograms of the children

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CHAIRMAN Now let us turn to the use of the Average Evoked Response of the cortex in the study of the hearing of infants and to the possibility that it might be made the basis of a definitive test of hearing that can be applied during the first weeks of life Dr Barnet and Dr Appleby have both prepared reports on this subject

## *Average Evoked Electroencephalographic Responses to Clicks in the Infant*

BARNET We have been studying the development of EEG responses to clicks and lights in a longitudinal and cross sectional population of normal infants We have been trying to relate the response not only to the infant's age, but to his state of consciousness, his type of prior sensory experience and other parameters of development We have also recorded the responses of a smaller group of infants with various sensory and neurologic deficits

As a sound stimulus we have been using clicks repeated at a slow rate, either 1/sec or 1/2 5 sec These have been delivered using an amplified one msec pulse output of a 1662 Tektronix wave form generator fed to an 8 inch loudspeaker 30 inches in front of the baby's head The loudest click is about 65 dB above adult waking threshold The duration of the peak intensity of the click is about 7 msec ,[1] frequency range, measured by a "Soundscope" frequency analyzer, is 75 to 2400 cps with a peak range at 600 800 cps

In a typical run responses at several intensity levels are recorded but we have not as yet varied the frequency or patterning of the sound

[1] This approximate value was determined by recording a set of 250 clicks on magnetic tape and using the computer to average and the X-Y plotter to display the averaged wave fo r Th r ethod was also used to check the simultaneity of the trigger and the stimulus

Evoked EEG responses to clicks are present in the early neonatal period in both full term and premature infants Their demonstration is greatly facilitated by using an electronic averager

In the experiments I will describe, the EEG amplified with Tektronix equipment was recorded on magnetic tape and later played back through an analog-digital converter (designed by Mr Walter Kropfl) to the Packard-Bell 250 general purpose digital computer which was programmed to average voltages at specified intervals through all or part of the interstimulus period, and to repeat the process until the desired number of averaged responses had been obtained The averaged response was displayed using an X–Y plotter A paper tracing was obtained by playing the tape through a modified Grass polygraph

I will use the first slide to illustrate a study done by Mrs Rhoda Goodwin and myself on click responses in the neonatal period Figure 26

This study was designed to examine the relationship between stimulus intensity, evoked responses, behavior, and heart rate in the normal human newborn Sets of 250 clicks were presented to twelve 2, 3, and 4 day-old infants, two boys and two girls at each age The infants were full-term with birth weights of 6 pounds or over and were products of uncomplicated pregnancy and delivery They had normal initial pediatric examinations and course in the nursery

The baby, lying on his back in his own bassinet, was placed in a small, electrically shielded enclosure, which was located in a small, dimly lit room in the newborn nursery suite The ambient noise level was about 55 dB Figure 26 illustrates a complete run for one of the subjects

Bipolar recordings from scalp electrodes applied with salt paste and bentonite according to the 10–20 International Electrode Placement System were obtained from left and right temporal central electrodes ($T_5 - C$ $T_6 - C$), and from a central-frontal electrode pair ($C_z - F_3$) The right mastoid was used for a ground electrode Heart rate was recorded with a precordial electrode

After an initial 5 minute period of recording without stimulus, a train of 250 unattenuated clicks at 1/sec was presented After a 2-minute interval without stimulation another train of 250 clicks at 1/sec, attenuated 5 dB from the preceding set was presented This was repeated until an attenuator setting of 30 was reached, at which time the sound was increased in intensity in 10 dB steps until attenuator setting 0 was again reached A period of no stimulation was recorded again at the end The recording period was about 90 minutes Three babies were presented with the same stimuli in reverse order, from soft to loud to soft, for an additional three babies the 0 attenuator setting was used throughout the run

Recording was done only when the baby appeared to be asleep, i e, when he lay quietly with closed eyes Observations of the baby's behavior were made in detail by marking a protocol designed for this purpose during the last 30 seconds immediately preceding the stimulus, then during the

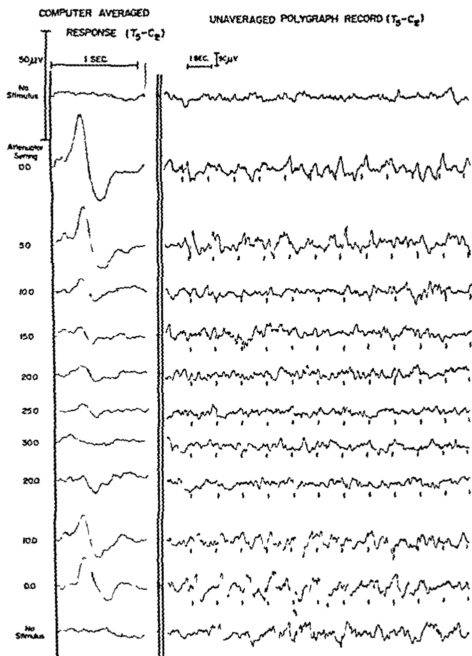



FIG. IV. 26. Computer averaged click evoked responses and the unaveraged polygraph record for the same lead are shown for a 2 day old subject. Descending-ascending order of stimulus intensity. Attenuator setting 0.0 is about 65 dB above adult waking threshold, attenuator setting 30.0 about 35 dB above. Note evoked responses in unaveraged polygraph rec[illegible] intensities.

*Acta oto lar.*

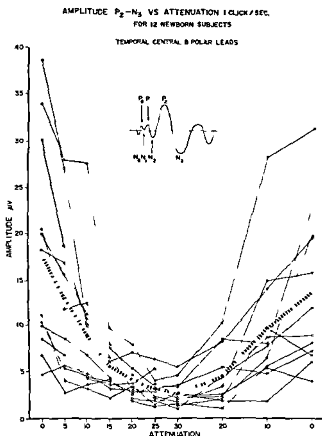



FIG IV 27 Amplitude of averaged click evoked response of 12 newborns vs changing attenuation Dotted line is average for all subjects Amplitude measures refer to $P_2$-$N_3$ Insert shows a typical averaged wave Upward deflection denotes positivity at $C_z$ with respect to $T_5$

first 30 clicks, the last 30 clicks, and the 30 seconds after the click was turned off All observable behavior was recorded, and a note made of that which seemed to be in response to the stimulus Head position was not controlled, but was noted

All of the babies studied showed evoked responses of a characteristic wave form The click response at a stimulus intensity of about 65 dB above adult threshold (attenuator setting 0) usually consisted of an initial small negative deflection followed by two, or more often by one, positive deflection ($P_1$) with an average latency of 95 msec and amplitude of 2 5 microvolts ($\mu$V) The largest component of the response (designated by us, $P_2$-$N_3$) showed an average peak latency (for $P_2$) of 267 msec and an average peak to-trough amplitude of 19 $\mu$V This component could sometimes be identified in the polygraph tracing

As the intensity of the stimulus decreases, the size of the $P_2$ component decreases With increasing intensity the size of response again increases

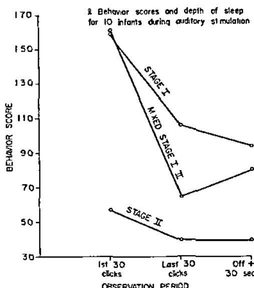



FIG IV 28 Average behavior scores and sleep stage vs observation period for 10 infants in descending–ascending series of auditory stimulation A high score denotes more overt behavior Infants in stage I (lighter sleep) show the most overt behavior, infants in stage II (deeper sleep) show the least There is more overt behavior during the first 30 clicks than during the last 30 clicks or the 30 seconds post stimulus

The earlier components of the response do not appear to be affected by stimulus intensity in the same manner as the later components They are more complex in form at higher levels of stimulus intensity and in those subjects whose responses are of high amplitude

Figure 27 shows the relationship of the $P_2 - N_3$ component to intensity for the 12 subjects These 12 and the 3 presented with stimuli in reverse order—from soft to loud to soft—showed unequivocal averaged evoked responses to sounds ranging from 65 dB above adult waking threshold to 45 dB (attenuator setting 20) At attenuator 25, two apparently normal infants showed no discernible response, and at attenuator 30, four of the fifteen infants showed no response The intensity-amplitude relationship is significant beyond the 0 001 level The $t$ test also showed that there was no significant difference in amplitude between the first and second presentation of the same intensity

### *Behavior and EEG Sleep Patterns*

Detailed behavioral observations were made as described above, and a score for total behavior for a given observation period was obtained Since the stimuli seemed to disturb the infants hardly at all, designating a behavioral change as a definite response to sound stimulus was difficult except in the very few cases when a slight startle or eye blink was observed during the first few clicks All observable activity such as eye, hand, or mouth movements, startling, or forehead wrinkling were therefore

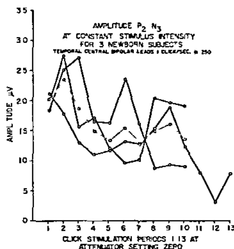



FIG IV 29 Amplitude of averaged click evoked responses of 3 newborns for successive sets of clicks at constant intensity (65 dB) N = 250 for each set Dotted line indicates the average for the 3 subjects

given equal weight in the scoring Analysis of variance of the ten infants in Group I, for whom complete behavioral data were available, shows significantly ($p < 05$) more behavioral activity during the first 30 clicks at each decibel level than during the last 30 clicks or during the off period immediately following

There was no more total activity with the loudest than with the softest click, but behavior that seemed clearly related to the clicks such as a slight startle reaction or a 1/sec eye blink was confined to the louder stimuli The amount of overt behavioral response to the stimuli was also related to the depth of sleep as judged from the EEG The polygraph record for each infant was scored for a sleep stage by a system modified from Roffwarg *et al* (1963), stage I being low voltage activity which we distinguished from the waking record by the relative absence of muscle artifact, and stage II being of higher voltage than stage I and sometimes having short bursts of 14–16 per second spindles

The relationship between behavioral activity, depth of sleep and the click period is shown in Fig 28 The larger number on the Y-axis indicates more activity There is greater activity during light sleep and during the first 30 clicks than during deep sleep, although a slight behavioral response is also seen during deep sleep

Statistical correlations were computed to examine the relationship between average evoked response characteristics, depth of sleep and behavior These show that deep sleep, as indicated by behavior and EEG, is associated with higher evoked response amplitude and latency.

Runs of low voltage fast asynchronous activity, especially at the beginning of the stimulus period, were characteristic of the polygraph tracing

for six infants This flattening of the tracing was sometimes preceded by a K complex Five out of six of these babies had smaller than average evoked response amplitudes It was thought that these infants might prove to be distinguishable from the rest of the group on the basis of age or behavior, but this did not prove to be the case

No significant differences in evoked response or behavioral activity were related to the sex or the age of the 2–4 day old infants

Evoked response characteristics at a constant stimulus intensity of 65 dB were examined for three infants (Fig 29) The amplitude of $P_2-N_3$ for the temporal central leads shows no significant correlation with time period although a downward trend in amplitude of response with time is evident ($K = +\ 321$, with significance beyond the 0 10 level ) The remarkably regular response increment or decrement seen with intensity change is not characteristic of the response amplitude-time relationship

To one of the above infants four additional sets of 250 clicks at the same intensity were presented A decline in response amplitude is evident as the number of sets of stimuli goes up

*Heart Rate*

Heart rate was counted for 5, 10, and 30 seconds before, at the beginning, and at the end of each stimulus period, and when the click was turned off, for the eleven infants on whom complete heart rate data were available Thirty second counts were used for analysis because detailed observations of behavior were being made for the same period The greatest cardiac acceleration from prestimulus level occurred when there was a startle response or a general body stirring Depth of sleep as judged by the EEG appeared to be related to the degree of cardiac responsiveness In Fig 30, cardiac rate changes from the 30 second period immediately preceding stimulation are plotted separately for the periods when the infant was in sleep stage I before and during the first 30 clicks and stage II before and during the first 30 clicks Rate changes when sleep patterns were mixed fell between those seen for stage I and stage II The largest acceleration of any observed, however, occurred with an infant who was in stage II of EEG sleep but who startled repeatedly with the loudest click

The responses were highly variable We could not demonstrate a statistically significant relationship between amount or direction of heart rate change and intensity of stimulus In general the infant's responses corresponded to the dictum which states that cardiac response to stimulation is a function of the rate prior to stimulation (Lacey, 1956, Wilder, 19[illegible]8 With high prestimulus rates, the infants tended to show less accelera[illegible] [illegible] even deceleration while with lower prestimulus rates, acceleration w[illegible] [illegible] this tends to be greater

[illegible] component of the auditory evoked potential of newborns occ[illegible] [illegible] 270 msec post stimulus It appears to have many of the char[illegible] K or vertex wave (Davis *et al*, 1939, Roth *et al*, 1956)

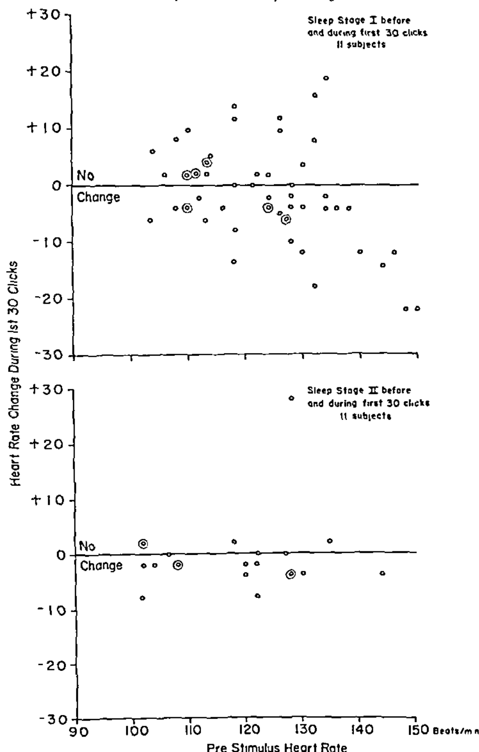



FIG 1S 30 Heart rate changes with click stimuli, regardless of intensity, are plotted sepa ely for two EEG stages of sleep There appears to be a narrower range of cardiac resp n e during deeper sleep although some infants when in stage I also show little cha in cardiac rate The one large cardiac acceleration seen during deep EEG sleep occ ed in a baby who startled repeatedly with the loudest click

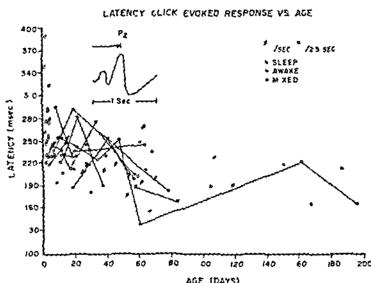



Fig IV 31 Decrease in latency of $P_2$ with age Points for the same subject are joined During the 1st 15 days but not later latency is also longer with the shorter interstimulus interval

in distribution and in its relation to stimulus intensity and state of the subject In the adult, however, the response latency for the *K* wave is in the range of 169–190 msec (Davis, 1964, H L Williams *et al*, 1962) Figure 31 shows the changes with age in latency of response to a 65 dB stimulus in a group of 60 infants Points for the same baby are joined There is a great deal of variability in response latency but the general trend is downward Part, but not all of the variability can be accounted

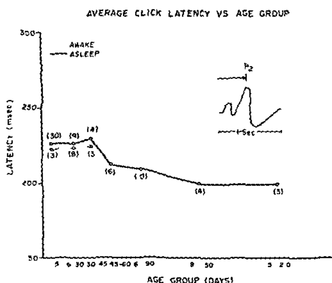



Fig IV 3 Average latencies of infants responses during sleep and wakefulness The number of individual records averaged for each age group is indicated

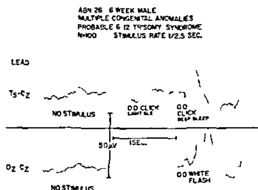



FIG IV 33 Absent click responses in infant with 13 15 trisomy syndrome and defects including absent tympanic membranes

for by considering the state of the infant Responses during sleep are of longer latency than responses when the subject is awake In Fig 32 the response latencies are averaged for age groups Response latency seems to remain constant or increases slightly for about six weeks and then begins to decline Response amplitude is more irregular, but there is a tendency for it to increase in the early weeks and then decrease Adult response amplitude under these recording conditions is between 10 and 20 $\mu$V

Figures 33 and 34 illustrate the use of the evoked response technique in the differential diagnosis of deafness in the infant Both babies had multiple congenital anomalies, due to chromosomal trisomy, deafness was suspected in each

*ABN 26, 6 week male*

This infant was born at term with a birth weight of 6 lb 3 oz to a 25 year old mother Multiple anomalies were noted at birth or soon after These included microcephaly with small fontanelle, a cardiac defect, probably tetralogy of Fallot, duodenal atresia, and non functioning left kidney He also had left enophthamos and ptosis, deformed low set pinnae with narrowed external auditory canals, and a hypoplastic mandible The diagnosis of Treacher Collins syndrome was entertained He was extremely hypotonic and the Moro response was absent Behaviorally he showed no clear response to either light or sound stimuli Chromosomal study showed a 6–12 trisomy

The evoked potentials of this infant are shown in Fig 33 Their presence suggests that this infant's lack of response to sensory stimuli is not on the basis of peripheral defect and implies intactness of at least part of the afferent pathway to the cortex

*ABN 20 2 day female*

This 2 day old female was born to a 40 year old mother at 36 weeks gestation with a birth weight of 4 lb 13 oz Multiple anomalies were evident, including

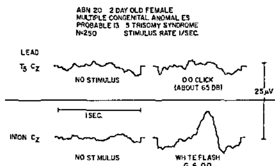



FIG IV 34 Click and flash evoked responses in infant with 6–12 trisomy syndrome

microcephaly with midline scalp defects, cleft lip and palate, hypoplastic left leg, ulnar deviation of left wrist, and a cardiac defect (Tetralogy of Fallot) The ears were low set and the auditory canals were narrowed The clinical impression was probable 13–15 trisomy. A Moro response was seen when the infant's shoulders were raised from the bed and then suddenly dropped, but no response to sound was elicited

The child succumbed at 10 days of age to the cardiac abnormality Autopsy revealed absence of the tympanic membranes Unfortunately the brain was not examined

This infant shows no EEG response to clicks, but a clear flash evoked response, of very long latency, is present (Fig. 34)

*Discussion*

The evoked response technique appears to be of value in the detection and differential diagnosis of deafness in the infant Responses at a low intensity of stimulation were detectable in most babies and modification of the technique such as using a longer interstimulus interval or irregular spacing would probably increase the reliability near threshold Varying the frequency of the stimulus would of course increase the usefulness of the technique in audiometry (See H Davis and S Appleby, this symposium ) Since the response is larger in amplitude during deep sleep, the diagnostic run in the infant should include recording during deep sleep

Considerable intersubject variation in both size and latency of the evoked response was found in normal subjects of the same age which did not appear to be correlated with sex or heart rate response Larger amplitude and longer response latency were often associated with deeper sleep, but there were large variations among individuals who were apparently in the same EEG stage of sleep, individual differences in the average evoked response cannot be explained solely on this basis

Since the characteristics of the evoked response change not only with age and state of consciousness but also with parameters of recording, sufficient data on normal subjects using a standardized procedure must

be acquired to insure the validity of the technique in diagnostic audiometry in the clinical setting

*Summary*

Normal sleeping newborn infants were presented with sets of clicks repeated at 1/sec or 1/2 5 sec In one study, for a group of twelve 2, 3, and 4 day old infants, intensity was attenuated in 5 dB steps from about 65 dB to about 35 dB above adult waking threshold, and then increased in steps to the original level To three infants, clicks were presented in the opposite order, i e from soft to loud followed by loud to soft Three other newborns were presented sets of clicks at a constant 65 dB intensity EEG responses to the stimuli were electronically averaged Temporal-central and central-frontal bipolar recordings were made Behavioral and cardiac responses were also recorded

1 The amplitude of the large late component of the averaged evoked response is a linear function of stimulus intensity This component is probably the K complex described by Davis It was discernible in all subjects to about 45 dB above adult threshold and in 11 of 15 subjects at 35 dB The amplitude-intensity relationship is seen in temporal central and central frontal leads The click evoked response of the newborn is of longer latency than the adult evoked response The amplitude and latency of the earlier components of the evoked response do not appear to be linear functions of stimulus intensity

2 For the click attenuation series, significantly more behavioral activity was observed during the first 30 clicks at each intensity level than during the last 30 clicks or the pre- and post-stimulus periods

3 When the EEG records were characterized by relatively high voltage and spindling, i e deeper sleep, less overt behavior was seen and cardiac rate responses to the stimuli were less evident

4 Under conditions of unvarying stimulus, the amplitude of response in temporal-central leads waxes and wanes, but over a period of about an hour (2500 clicks) does not significantly decline in amplitude although a tendency to decline begins to emerge However, response amplitude in central-frontal lead decreases over the same time

5 At a stimulus presentation rate of 1/sec, the amplitude of the evoked response of the sleeping newborn is determined largely by the intensity of the stimulus Depth of sleep, however, also influences the response, i e deep sleep is associated with larger responses of longer latency than lighter sleep

Data on the changes in amplitude and latency of the evoked response with age is presented for a group of 60 babies

Several examples of the responses of abnormal young infants, especially those with defects of the auditory system, are presented

It is concluded that the EEG evoked response technique is of value in the detection and differential diagnosis of deafness in infancy

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## *The Slow Vertex Maximal Sound Evoked Response in Infants*

Appleby At the Toronto General Hospital we are studying the slow non specific sound evoked response in newborn infants Preliminary work with normal and hard of hearing adults indicated that the response can be obtained easily and that this response gives an estimate of the auditory threshold (Appleby *et al* 1963) This is the report of our experience using the Computer of Average Transients in over 160 newborn infants We have found this procedure can be applied to the very young infant and that it gives an indication of hearing in these small people

The babies studied have been from the newborn nursery at Toronto General Hospital and were from two to ten days old Because we were interested in determining the response in normal infants only babies with a birthweight over six pounds were tested No babies anoxic at birth or with significant jaundice were tested No babies with a history of hereditary deafness were encountered in this series Babies whose mothers had had rubella during pregnancy and babies with known congenital defects were excluded

The first 35 babies examined were tested with clicks If the infant was asleep the response was easily obtained but in the waking state the results were equivocal This we have attributed to movement artefact

Clicks unfortunately are unsuitable for quantitative work since their frequency spectra and intensity cannot be measured accurately To overcome this we next used pure tones from a standard audiometer However these because of their slow rise time were not as effective in evoking a response These problems we took to Dr Hallowell Davis who suggested as an effective compromise narrow band tone pips We have used his method for the remainder of our study

The characteristics of the test tone are important The tone pip produced

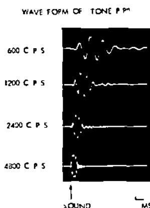



Fig IV 35

by our equipment has a rapid rise time and a known frequency Moreover, the intensity can be determined by having an experienced observer balance the loudness of the tone pip with the tone of a pure tone audiometer The loudness of the tone pip can be altered in dB steps with the attenuator

Fig 35 shows the wave form of the tone pip produced by our equipment

Fig 36 is a diagram of the apparatus we are now using The stimulator is the pacemaker for all the equipment It simultaneously induces the sound stimulus, starts the analyzing and averaging sweep of the computer and the monitoring sweep of the oscilloscope and polygraph To produce the sound, the square wave from the stimulator rings the passive filter which has been tuned to the desired frequency The signal from the filter passes through a calibrated attenuator which gives the required intensity and then into the speaker amplifier The electrical activity from the scalp electrodes on the subject's head passes through a two-stage amplifying system, at present we are using a model 5 Grass polygraph The amplified signal then goes to the computer which analyzes, integrates and displays the averaged evoked response which is photographed Throughout the test, the activity from the scalp is recorded as an EEG

Fig 37 is a photograph of the equipment we are now using

At first the testing was done in an examining room on the ward with

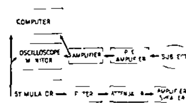



Fig IV 36

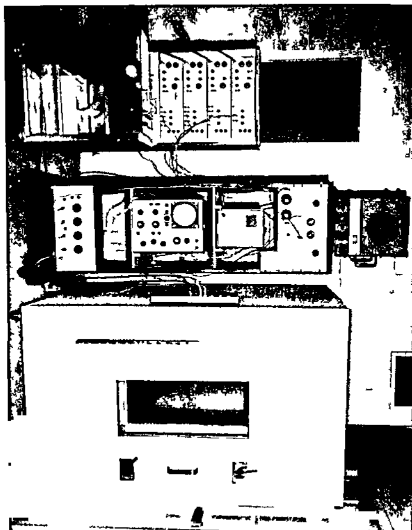


Fig IV 37

the baby lying in its bassinette By selecting a quiet time of the day, it was sometimes possible to get the ambient noise down to 50 or 60 dB The final fifty babies have been studied in an acoustically insulated chamber This cubicle is double walled and provides about 25 dB of sound attenuation It is electrically shielded and is sufficiently large to accommodate an infant s bassinette or a sitting adult, and is sufficiently small to be pseudo portable At least it can be moved from one area of the hospital to another

An idealized response, as recorded from the vertex is shown in Fig 38 Some of the peaks may not be as clearly defined in individual tracings but the overall pattern is similar to that reported by P A Davis (1939), H Davis (1939), Abe (1954), and Derbyshire (1958) Usually a prominent vertex positive peak appears at about 125 msec and a vertex negative peak at about 250 msec There is variation in the latencies from individual to

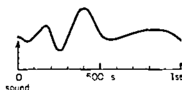



FIG IV 38

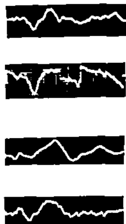
individual (Fig 39) One of the factors that decreases the latency of the peaks is an increase in the intensity of the sound stimulus (Appleby 1963 Abe 1954) Peaks with longer latencies are seen in the second half second but the time of their occurrence is more variable

A typical cortical evoked response shown in Fig 40 was subjected to a Fourier Analysis The result of the cosine analysis is shown in the lower curves of Fig 40 The principal frequencies are below 3 cps and the analysis showed that less than 5% of the energy appears in frequencies above 10 cps

Fig 41 illustrates the responses obtained when recordings were made simultaneously from the vertex to the ear from the forehead to the ear and from the occiput to the ear Recording from the forehead the peaks are still clear but are of lower amplitude and of longer latency The response is very small and scarcely apparent when recording from the occiput

To determine the effect of pitch pips of 600 1200 2400 and 4800 cps were used as stimuli These were all effective in eliciting the response (Fig 42)

AVERAGE OF 30 RESPONSES TO 2400 C P S
STIMULUS 4 DIFFERENT BABIES



1 SEC

FIG IV 39

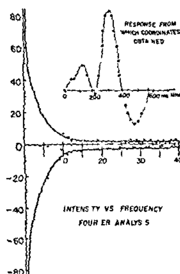



FIG IV 40

The sound pressure level necessary to obtain the response in the sleeping infant was studied in forty babies Tone pips in the 1200 cps range were presented at three second intervals Fifty responses were averaged With a standard amplification and using the peaks having 125 and 250 msec latency, a peak to peak height of 3 mm or more was regarded as a positive response Using this arbitrary standard, the peaks could be easily identified A positive response was obtained in all babies at 60 dB and in 85% at 50 dB At 40 dB, a positive response was obtained in less than 40% of the trials At less than 40 dB, the results were unreliable

In determining the threshold, the order in which the sounds are presented is a factor Although we have not sufficient data for a firm statistical analysis, the trend appears to be similar to standard audiometry, if the louder sound is presented first, the threshold is lower

The optimum interval between stimuli was investigated In general it was found that the greater the time between stimuli the larger the response This applied especially to the later components of the response Fig 43

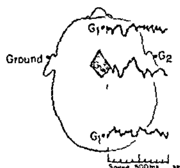



FIG IV 41

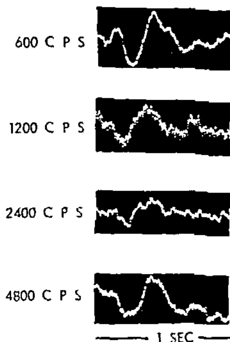



Fig IV 42

shows a gradual increase in the size of the response when the frequency of stimulation is decreased

Habituation or fatigue was not observed during the first two hundred stimuli at any of the rates tested

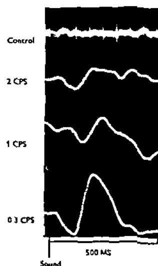



Fig IV 43

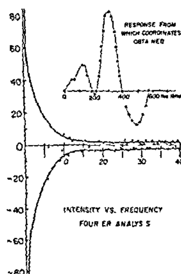



FIG IV 40

The sound pressure level necessary to obtain the response in the sleeping infant was studied in forty babies Tone pips in the 1200 cps range were presented at three-second intervals Fifty responses were averaged With a standard amplification and using the peaks having 125 and 250 msec latency, a peak to peak height of 3 mm or more was regarded as a positive response Using this arbitrary standard, the peaks could be easily identified A positive response was obtained in all babies at 60 dB and in 85% at 50 dB At 40 dB, a positive response was obtained in less than 40% of the trials At less than 40 dB, the results were unreliable

In determining the threshold, the order in which the sounds are presented is a factor Although we have not sufficient data for a firm statistical analysis, the trend appears to be similar to standard audiometry, if the louder sound is presented first, the threshold is lower

The optimum interval between stimuli was investigated In general it was found that the greater the time between stimuli, the larger the response This applied especially to the later components of the response Fig 43

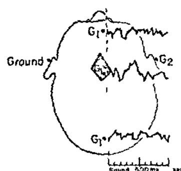



FIG IV 41

conditioning situation This Contingent Negative Variation (CNV) reflects the probability of signal association as estimated by the subject and is independent of stimulus amplitude or energy It is particularly large and consistent when the stimuli are purely semantic, provided that they are meaningful to the subject and that some decision or action is required

In young children the CNV is small and variable but can be augmented by social influences such as persuasion, instruction, admonition, and competition.

This effect has been investigated in relation to the human auditory system, bearing in mind that in normal conscious people it is impossible and unrealistic to consider perception in any one modality in isolation The CNV is a clear indicator of the cerebral mechanisms tending to link signals from all sources in significant patterns for effective action

The independence of the CNV on the intensity of stimuli is seen most clearly in situations when an auditory conditional signal is reduced to threshold, the CNV persists at full size as long as the conditional event is perceptible. Furthermore, a "negative" stimulus can be as effective as a positive one For example, the interruption of a gentle continuous tone may be used as a conditional stimulus, followed by a visual or tactile imperative stimulus one second later The *cessation* of the auditory stimulus then evokes a primary response in the nonspecific frontal cortex, followed by a CNV leading up to the imperative response exactly as when the conditional stimulus is a click or tone An interesting feature of responses to "negative" stimuli is that the latency of the primary components is much longer than for positive stimuli (150 msec to peak compared with 100 msec ) although the features of the CNV are identical The existence of large responses to very faint or "negative" auditory stimuli is not really surprising since auditory communication is not dependent on mere amplitude and the pauses and spaces between sounds are as significant as the sounds themselves The recognition of this fact is, however, an important development in the understanding of auditory physiology since hitherto the emphasis has been on the energetics rather than the semantics of the stimulus response characteristics It is now clear that the sensory projections to the frontal cortex operate not as simple transmission channels but as significance discriminators with a very efficient "automatic gain control" that minimizes the effects of mere intensity variation

It is interesting to consider the responses to auditory stimuli in nonspecific cortex over the whole range of associational situations At one extreme is "classical" conditioning using, for example, the unconditional blink to mechanical stimulation of the cornea, preceded by an auditory signal At the other is the presentation of a purely semantic signal such as an interesting picture or problem, in which there is no energy, a high degree of subjective significance, and no motor response Between these extremes is "Operant" conditioning in which the auditory signal is followed by another stimulus to which the subject is instructed to respond by some

suitable action In the "classical" case of defensive conditioning the primary non-specific auditory response to a conditional stimulus consists mainly of a single large negative wave with a very small secondary component (apart from the CNV) In operant conditioning the second component is usually more prominent, and in the special case of a "negative" stimulus there is no primary component at all and the only feature is the secondary response with the long latency already mentioned In the case of purely semantic conditioning the secondary wave is usually as large as the primary one and there are sometimes several secondary waves leading up to the CNV There seems a possibility that these two components of the auditory responses in non-specific cortex may represent two physiological mechanisms, the first, with a short latency and arising only with positive stimuli is an on-effect, projected directly from the anterior thalamic nuclei to cortex, the second, appearing with positive semantic or negative stimuli, with a longer latency may represent transmission from the specific sensory cortical areas through the long commissures to the anterior non specific regions

In these terms it would seem possible to distinguish between three processes The first is the diffuse projection of positive sensory information—'something has happened" This is the brief early non-specific response which habituates slowly during monotonous stimulation and is essentially an "on effect" The second is the later brief response, which may be multiple, and appears most clearly with negative stimuli during semantic conditioning The third is the CNV which links the conditional responses with subsequent events as a prelude to action

The dependence of the secondary evoked components, especially the CNV, on the statistical information content of the signals, is most clearly seen during trials of extinction and equivocation, that is, when only a proportion of the conditional stimuli are reinforced by association with imperative or important signals In these situations the amplitude of the CNV reflects very accurately the subjective probability of significant association In normal adult subjects the subjective and objective probabilities are well matched, the brain seems to perform a running average of associations extending back to 20 or 30 experiences In mentally disturbed patients and particularly in children the subjective and objective estimates of significance are often widely discrepant, but in normal children the divergence can be diminished by verbal explanation during the experiment Perhaps the most dramatic illustration of how closely the CNV is related to mental states is seen in hypnosis The primary evoked potentials are practically unaffected by hypnotic suggestion, but if a subject in deep trance is told, for example, that previously significant conditional clicks will be heard frequently without reinforcement the CNV linking the auditory and imperative responses subsides even when in fact all the clicks are duly reinforced

If these observations seem to emphasize the inaccuracies and vagaries *of the brain as a computer, we should recall that our sensory systems* have a social as well as a physical function, perhaps we can envisage the

development of a social physiology in which the interactions between brains can be considered as systematically as we now describe the interactions of brain regions

CHAIRMAN: We thank you, Dr Walter, for a most important new contribution to electroencephalography

WALTER The method of electrical evoked responses could very well be combined with the Peepshow technique because in the Peepshow you have a conditional stimulus, a visual presentation, an operative response, etc We have done a rather similar thing, although not in relation to deafness We obtain beautifully clear evoked responses that vary with the semantic content of the presentation This situation seems to me to promise a rather complex but nevertheless very clear and penetrating type of analysis, particularly if one looks for both the slow evoked response (ER) and the contingent negative variation (CNV)

CHAIRMAN Many of us are very optimistic about the possibility of determining the audiograms of very young children by means of cortical evoked responses An average response computer is a great help here if not an absolute necessity Unfortunately the instruments that most of us are using are rather expensive, and would be out of reach of most clinics and of private otologists or audiologists in general But the research instruments that we are using may be both more complicated and more versatile than would be necessary for simple routine audiometry It is therefore very important to ask whether it may be possible to design a simple, inexpensive but adequate test system that might make this new method generally available Dr Scott, I believe you have developed such an instrument

### *An Inexpensive Averaging Computer*

SCOTT I and my colleague, Mr W V Weiss, explored the possibility of building an economical instrument to extract the sound evoked delayed cortical response from the EEG Versatility is not required but it must be reliable and accurate and also simple to operate We undertook to assemble it from stock commercial parts

The block diagrams of the inexpensive model will be the same as the block diagrams of the equipment developed by Hallowell Davis that we have been using, but components having very little or no versatility have been selected to perform specific limited tasks

In order to specify the characteristics of the computer a typical evoked response curve was selected and subjected to a cosine Fourier Analysis. This showed that the frequencies over 10 cps contributed less than 5% of the total energy Consequently 20 ordinates for a one-second analysis

would give a reliable curve and by increasing the number of ordinates to 50 a reliable curve for frequencies up to 25 cps is obtained

A square-wave generator will act as a master clock, triggering the computer whenever it emits a pulse to the passive filters It will produce square-wave pulses of 1 msec duration at 6 volts at intervals ranging from 125 msecs to 10 seconds

The square wave will enter a passive filter which can be set to pass *600, 1200, 2400, or 4800 cps The output* will be at least 30 dB down per octave on each side of the center frequency The output, which is now a "tone pip", will enter an audio power amplifier with a flat response between 20 and 15,000 cps The output of the power amplifier can be attenuated in 1 dB steps from 0 to 120 dB The pips are then fed to a loudspeaker or earphones

The cortical evoked potential is observed by examining the EEG, recorded between the vertex and an ear lobe, for a period of up to 1 second after each stimulus The periods of analysis selected are either 1 second, $^1/_2$ second, 250 msecs or 125 msecs The amplified EEG is fed to an electrical analog computer The timing and control circuits of the computer provide a series of pulses which operate a device similar to a telephone stepping switch but faster This will first close the signal relay and then the storage capacitor relay #1, then it will close the signal relay again and then storage capacitor relay #2, and it will repeat this procedure for all 50 storage capacitors In this way the amplifier samples the incoming, amplified EEG signal at 50 points in time It holds the sampled wave shape in its capacitor memory, and automatically adds the next wave algebraically to its previous contents Thus it performs an averaging function Relays were selected to perform the operational switching because the shortest dwell-time (2–5 msec ) is within the capability of an electromechanical device and the relay switches have an open circuit resistance greater than $5 \times 10^{11}$ (ten to the eleventh power) ohms This high resistance is needed to prevent leakage from the storage capacitors

Fifty responses will be averaged as a routine

The memory capacitors are sampled by the same control circuit The stored voltages can be displayed on a cathode ray oscilloscope, and measured, traced or photographed as desired

Using standard components, a commercially available stimulator, a 3-inch cathode ray oscilloscope and regulated power supplies, the cost of the components is about $1800 00 An additional $500 00 would provide for cabinets, wire, terminal blocks, etc and construction cost Thus the total cost would be $2300 00 The only additional unit needed is a single channel EEG amplifier A camera, if desired, would increase the cost somewhat more

EDITOR (After the conference Dr Geary McCandless, whose work was cited by Dr Røjskjaer in his summary of the Copenhagen conference, was invited to contribute a note describing his original instrument and also to

state his opinions as to the specifications for a clinical testing unit The following is his evoked response )

McCANDLESS *Discussion of Simplified Devices for Clinical Testing by the Method of Auditory Evoked Responses*

In recent years a variety of computer systems has been developed to detect average evoked potentials, both in man and animals There are presently available a number of small biological digital computers for online determinations of average evoked responses In most cases, however, the cost of these units is prohibitive for use in a clinical setting Also, they do not contain a stimulus source and controls, a program circuit or auxiliary equipment which must be purchased separately

Prior to designing a clinical unit, careful determination of the effects of various stimulus parameters needs to be decided However, the following items should be considered in the design of any clinical device

1) The device should be sufficiently inexpensive as to permit purchase by hospitals, medical centers, and hearing centers

2) Its controls should be sufficiently simple to permit operation by clinic personnel or trained technicians

3) It should be a relatively small self contained unit exclusive of peripheral equipment such as oscilloscope or recorder

4) The unit should contain its own stimulus source and attenuating device

5) It should contain an accurate time base and self contained programming unit for determination of total number of stimuli, stimulus duration and repetition rate

6) Provision should be made for oscilloscopic monitoring on line of the average response as well as raw EEG data

7) It should contain sufficient gates so that the fastest component of the evoked response is easily definable, and yet few enough gates to reduce cost Assuming that one half cycle can be identified using three discrete points, a 50 channel unit with 10 ms gate widths could accept frequencies up to about 50 cycles per second Since the major portion of the evoked response is completed by 500 ms, 50 gates with 10 ms widths would be sufficient for most purposes

About four years ago, we designed and built a summing device with the above considerations in mind, but which had additional flexibility in terms of stimulus control Another objective of the original unit was to produce a device which consisted of conventional, easily-maintained circuitry which could be operated by a trained technician It was constructed as a single unit (19 inches × 20 inches × 23 inches) and contained stimulus controls, program circuit, memory circuit and power supply The auxiliary equipment included a strip chart recorder, oscilloscope and EEG preamplifier

In operation, silver disc electrodes were attached to the subject's head and potentials fed into a low level preamplifier with a gain of 20,000 to 100,000 For most studies the computer was set for gate widths of 10 ms,

making a total scanning time of 500 ms In principle, the method used was similar to that described by Dawson (1) where capacitors were charged, each at a fixed time with respect to the stimulus onset The system of switching from one capacitor to the next was patterned after a device described by Cox using glass reed switches (2) The fixed intervals were set by utilizing a time base set by a 1600 cycle tuning fork oscillator By using nothing but digital and logic type circuits to establish the time intervals for all functions, no calibration was required and highly reproducible steps over a considerable range could be achieved by means of simple selector switches connected to cathodes of dekatron selector tubes The time intervals for stimulus repetition rate, stimulus duration and total epoch length were determined by this means With this unit, clear evoked responses could be seen in as few as five to ten repetitions However, fifty stimuli were used routinely for most subjects

The charges stored on the capacitors could be retained until readout could be made and the readout system was not destructive The averaged response could be displayed on an oscilloscope or read out on a strip chart recorder for later viewing

Although our original unit used a method of mechanical switching from one capacitor to the next, there appears to be some virtue in a system with electronic switching The more rapid electronic switching would permit more time per gate for data acquisition

There are also advantages in a device utilizing magnetic core memory However, this method of memory storage requires that the data be converted from analogue to digital form The prototype utilizing electronic switching and magnetic core memory is presently being designed specifically for clinical application, and the total cost is estimated to be around $3000 00

*References*

1 Dawson G D, 1950 Cerebral responses to nerve stimulation in man *Brit Med Bull* 6 326

2 Cox R R, and Evarts, E V, 1961 An evoked response detector, *Electroenceph Clin Neurophysiol*, 13 478

Walter The average response computer that we used originally employed a storage system which we built ourselves To develop a similar one commercially would cost about $500 00 as a basic price A two-channel averager of our type should cost less than $1,000 00

Davis In other words if we know exactly what we want and our demands are not too great, a relatively simple computer can do the job and the price can be kept down within limits that would make it practical as a clinical instrument

WALTER: Commercial equipment that is generally available at present has a much larger storage capacity than is necessary. In physiological applications we don't ordinarily use more than one-tenth of the storage capacity of these commercial instruments, beautiful instruments though they are. The large capacity is why they are much more expensive than they need to be.

DAVIS: Our own digital data processing instrument, which was developed for research, is much more expensive and elaborate than is required for routine testing. We built it to be elaborate and versatile because we had not yet found out exactly what it is that we need for the clinical application.

DAVIS (afterthought): One more word about the acoustic stimuli, the tone pips. We have found these "filtered clicks" very useful because they are definite in time and therefore evoke clear time locked responses. They also have a good deal of tonality, although they are far from being musical tones. They sound like thuds, knocks, taps, snaps, and clicks. The electrical circuitry necessary to generate them is simple and relatively inexpensive. So far very good, but we are now finding that the particular rise-time and duration that we have been using may not be ideal. Too much acoustic energy is scattered up and down the scale. The result is that we cannot *map accurately an abrupt high-tone loss or "tonal gap". Our acoustic probe* is too blunt. As it happens, these abrupt audiograms appear rather frequently among our children and give the familiar clinical pattern of apparent good hearing but failure to understand and learn speech. We are about to try a different form of tone pip with a slower rise and fall. It will be much closer to a very brief musical note. My point is that I do not think we are quite ready to freeze the specifications on the acoustic stimuli for the proposed clinical instrument, except perhaps for testing infants.

CHAIRMAN: For the benefit of those who are not familiar with electro*encephalography and evoked responses I will emphasize that we have now* discussed three different electrical responses that can be recorded from the human scalp and for which the average response computer technique is appropriate. The first type of response is the myogenic reflex which Dr. Lowell illustrated. This response has a very short latency and seems to represent some primitive pattern of motor activity. The second type is the so-called "slow" non-specific response that most of us have been talking about. In fact everyone from Dr. Lowell to Dr. Walter showed at least one record of it, and Dr. Derbyshire clearly was able to see it in some of his unaveraged EEG tracings. The third type is a really exciting new one which Dr. Walter has described. This is a change of potential, a shift of the baseline, that occurs between a conditioned stimulus and an imperative stimulus.

This is particularly important because it allows us to look into the brain objectively, so to speak, at the level of meaningful associations

I will also point out that none of these three responses is the response of the primary auditory cortex in the temporal lobe The corresponding primary responses can be seen in the tactile and the visual systems but for reasons of anatomy, the physics of electrical volume conductors, and the confusion introduced by the reflex responses of the temporalis muscle, the primary auditory response is simply not available for study by external electrodes

WALTER If I may make a concluding comment I will say that the slow response is merely the sign of arrival of information at the cortex It tells you nothing about how this information can be used It does prove, of course, that the person's auditory apparatus is intact, but it does not prove that the person will be able to understand or can handle the incoming information

We have studied children down to the age of three The "expectancy wave" develops quite nicely in children of this age, but with decreasing age the importance of the social factors becomes greater and greater

### *Discussion of Presentations on Evoked Responses*

EDITOR'S NOTE Much of the discussion of the EEG papers is omitted because it consisted chiefly of minor questions of technique or else confirmatory remarks concerning similar observations that had been made in each speaker's own laboratory

DAVIS All of us who are recording average responses are well aware that in favorable subjects we can frequently see the individual responses With strong stimuli they may stand out very clearly indeed Dr. Derbyshire uses them as one of his criteria With the average response computer, for clinical use as an audiometric method, the problem is to get the greatest amount of voltage per minute from the subject because the time that the subject is available and cooperative is the bottleneck We have recently found that one stimulus per second actually seems to be the most efficient rate, although to get the largest single individual responses the interval should be about 10 seconds

### *Polarity of Evoked Response Records*

WALTER I wish to make an important technical point A lot of confusion has arisen today and will arise in the future about the polarity of the records of evoked responses In my own records an upward deflection always means that the active electrode is becoming more negative Dr. Davis, I believe you are using the opposite convention

DAVIS: That is true.

WALTER: The fact is that people who have machines which were developed by engineers are using positive-upward, while physiologists who have developed their own machines generally record negative upward because that is the classical physiological convention I believe that very soon we should all adopt a universal convention one way or the other It should not be restricted to this particular type of study but it should be world wide in application and we should all be bound by it Otherwise, enormous confusion can arise We are in the midst of such confusion right now We can see it again and again all through the literature The symposium on Sensory Evoked Responses in Man in the Annals of the New York Academy of Sciences illustrates it perfectly

GOLDSTEIN (in absentia) I agree that uniformity of polarity is desirable but I disagree that it is proper to achieve this uniformity by making negative up and positive down Since most people resort to cartesian coordinates for graphic representation of electrophysiologic and other phenomena, and since the first quadrant is implied in most graphs unless specifically noted otherwise, an upward deflection is the positive direction to the very large majority of workers in the biological and physical sciences The chief dissenters to this nearly universal practice are electrophysiologists, but even many electrophysiologists use the conventional cartesian directions in depicting evoked responses, as exemplified in "Sensory Evoked Response in Man", *Annals of the New York Academy of Sciences*, 1964, *112*, 1–546

We can achieve uniformity by standing on our heads Such a choice, however, would put us 180° out of phase with the majority of scientists in other fields, and with a substantial number of electrophysiologists By standing on our feet and regarding positive as up we make a more logical choice in terms of cartesian coordinates, we make ourselves more consistent with the large majority of scientists in all fields who already observe such a practice, and we shall become equally as consistent as a group as if we all regarded positive as down

DAVIS There has been double confusion in regard to these evoked responses because some of us have not been quite sure which was the active electrode Dr Derbyshire and I had some earlier correspondence as to whether the auditory response originated at the vertex or down by the ear But on the main point, Dr Walter, I agree with you completely and I am very glad that you have put this remark on the record We *must* do something about this

WALTER I hope that in the publication of this symposium the individual authors will all make very clear which convention they are using

## F. Summary

### *Retrospective Summary of Definitive Tests for Hearing in Young Children*

Chairman (to the readers of this volume) The following retrospective summary of all of the presentations and discussions of definitive tests of hearing in young children was kindly prepared by Dr. W. Grey Walter, who devoted the entire Saturday immediately after the conference to this task Dr Walter has done more than summarize, he has developed a logical organization He has grouped the tests and analyzed them from the physiological point of view He has introduced useful and stimulating after-thoughts, and he repeatedly suggests new experiments or important extensions of present studies Repeatedly he indicates where modern computer methods might bring the difficult and even the impossible within the reach of an experimenter Dr Walter properly devotes a considerable part of his summary to the cortical evoked responses properly because of his personal interests and special competence in electroencephalography and because the evoked electrical responses are both the newest and the most promising responses that are eligible as criteria for audiometry in very young children

### *Retrospective Summary, by W. Grey Walter*

Walter The various responses to sensory stimulation in man have been studied from the earliest days of physiology, but their characteristics in young children are still to be defined in detail In particular, the responses to auditory stimuli although qualitatively familiar, are difficult to quantify without quite elaborate instrumentation

#### *Startle Response*

Wedenberg has shown that even *before* birth loud sounds evoke two types of response in the foetus after the twenty-sixth week of gestation The first type is a primitive "startle" reaction, that is a generalized muscular contraction, mediated by quite primitive central nervous structures This does not involve the cerebral cortex and is superimposed on the background of spontaneous foetal movements This may require integrity of the cochlea but could also arise from stimulation of labyrinthine structures since Bickford has demonstrated short latency muscular responses to auditory stimulation in deaf subjects The intra uterine startle response is reported to diminish with repetition, i e to "habituate", and this feature could be used to determine whether cochlear "hearing" is involved, stimulation at one frequency, say 3 kc/s could be continued until the response had habituated and the stimulus could then be altered to say 1 kc/s If there

were any degree of cochlear discrimination, the response should reappear with the novel stimulus

The other type of response is a brief tachycardia followed by bradycardia The foetal pulse rate rises from about 135 per minute to as high as 160 and then falls to 80 This is a gross change which scarcely needs instrumental elaboration, but the effect of weaker sounds and extension to a crude pre natal audiometry might be achieved by use of the LINC program for foetal EKG identification, coupled to a mean zero type of cardiotachometer This would indicate very clearly deviations from the mean rate and should settle the question of whether 'hearing" is involved in a useful sense There are other instruments, developed specifically to follow the foetal heart rate through delivery, that might be employed

Similar responses can, of course, be observed in neonates, with increasing difficulty as the diversity and intensity of spontaneous activity increases For this reason the most convenient condition to study is sleep, which, as Wedenberg mentioned, is the normal state of babies during the first weeks of life During the first few weeks other responses to sounds appear, the blink reflex and respiratory arrest are common in waking babies and awakening from sleep is perhaps the easiest response to identify Wedenberg finds that a 3 kc/s tone at 75 dB will awaken normal babies within one minute Here again, audiometry of some sort could be envisaged, almost as a standard routine

*Orienting Reaction*

During the first year of life the startle response normally evolves into an orienting reaction which has been studied intensively by Russion neurophysiologists since it forms the basis of many Pavlovian studies This reaction obviously depends on stereophonic reception and its absence indicates unilateral hearing deficit The orienting reaction is accompanied by various autonomic responses—tachycardia blood pressure rise, limb and finger volume reduction and electrodermal responses All these effects require instrumentation and all are superimposed on "spontaneous' variations which demand careful statistical analysis This is a very serious difficulty in the application of autonomic response analysis to sensory loss studies Visuo-manual interpretation is tedious and misleading and this might prove a useful field for computer application The problem is very similar to others in the field of seismography and radio-astronomy and suitable programs may already by available Even simple averaging, as for the evoked brain responses to be described later, would be better than the unaided eye

*The Course of Maturation*

At this stage we can see only too clearly our profound ignorance of normal maturation The gross 'milestones" in human development are well recognized (Gesell), as, of course, is the pattern of physical growth (Tanner), but the only character of psychophysiological maturation which all

agree on is that it is enormously variable both from child to child and in one child from time to time Piaget has suggested that psychological development goes through various stages which he defines from the results of performance tests; he further suggests that the transition from stage to stage may be rather abrupt If this is so—and there are good theoretical reasons for supposing so—we are faced with a particularly awkward situation since the features of behavior on which we would like to base our decisions are not distributed "normally" in the child population and we must be very careful as to how we use traditional statistics The safest course is to rely on non-parametric methods in which normality of distribution need not be assumed These methods are not always compatible with simple measures such as arithmetic means and it would be interesting to have the opinion of experienced computer statisticians on these problems, the extraction of the harmonic rather than arithmetic mean might be more trustworthy since the time occupied by the events varies over a wide range

*The Electrodermal Response*

The electrodermal response of children to auditory stimuli has been studied for many years but with little practical success Known variously as the Galvanic Skin Response and the Psychogalvanic Response, this effect has two components a fall in ohmic resistance of the skin, and the generation of a small potential difference across the skin The resistance change is sometimes called the "Fere effect" and the potential change the "Tarchanov effect" The origin of neither is fully understood, but most experimenters agree that apparently both vasoconstriction and sudomotor activity are involved For this reason there is a high but not absolute correlation between the EDR and plethysmographic records of finger volume. In spite of the obscurity of its origin, the EDR has a surprisingly constant latency, as demonstrated by W Hardy for the Tarchanov effect, and this can be used to identify evoked from spontaneous changes Walter (1960) showed that in adults the latency of the Fere effect varied with the contextual position of the stimulus the latency to unconditional ("startling") stimuli averages about 1 5 sec while that to conditional or warning stimuli averages about 2 0 sec In children this differentiation is still possible, but, as might be expected the utility is in the opposite direction, that is, the latency of an EDR can help to indicate whether a child regards the stimulus as intrinsically, unconditionally arresting or only conditionally so in relation to some subsequent event The longer latency of conditional responses suggests a longer time and pathway in the CNS and this suggests some application to the study of comprehension in older children

*Conditioning Pavlovian and Operant*

The reference to conditional responses introduces the concept of using the [illegible] functions in the estimation of hearing loss A distinc[illegible] between so-called classical or Pavlovian condi-

tioning in which the conditional stimulus is associated with another stimulus having an unconditional (usually involuntary or unconscious) reflex response, and operant conditioning in which the subject performs some complex action related to the unconditional stimulus, usually following instruction In human experiments few "classical" conditioning situations are feasible, the eye blink is almost the only convenient reflex and this could certainly be investigated in relation to hearing loss, using an auditory stimulus as a *conditional* stimulus and a corneal stimulus as the unconditional one For such a study slowly rising tone pips would be suitable since they do not themselves evoke a blink reflex as clicks are liable to do

### The Peepshow

The most successful and straightforward form of operant conditioning in hearing studies is probably the peepshow system described by Dix and Stratton With this arrangement the child learns by instruction to press a button on hearing a test tone in order to reveal a picture or movie sequence This arrangement lends itself to several developments not only in pure tone audiometry but also in tests of comprehension, since words and phrases could be used as in play audiometry The peepshow could therefore become a sort of teaching machine if the exposure were to be significantly related to the conditional sound by way of the operant response As will be explained later, this arrangement could easily be combined also with contemporary methods of brain-response analysis to provide a multilateral study of conditional behavior and cerebral function as related to hearing

### EEG Audiometry

The systematic investigation of brain responses to auditory stimuli is a particularly important though relatively recent development For many years workers in Electroencephalography (EEG) have known that auditory stimuli are liable to affect the resting pattern of intrinsic rhythm In the waking state these changes are often hard to identify, particularly in children who show so much irregular activity over a wide frequency band There are several problems here, not least the classification of juvenile EEG records Many deaf children exhibit the peculiarities described by Bertrand but none of these is specific to deafness and some are seen in children without clinical signs or symptoms There is no doubt that *all* children suspected of hearing deficit should be studied carefully from the EEG standpoint with all available techniques of physiological and technical analysis including sleep and sensory stimulation in several modalities In this way cases of brain damage and gross sensory response anomalies can be detected at an early stage In older children the detailed analysis of the intrinsic rhythm patterns can help to indicate the predominance of modality preference which Silverman emphasized as one of the crucial factors in the appraisal of hearing potentiality As an example,

persistent alpha rhythms are usually associated with relative poverty of visual imagery and corresponding dependence on auditory, verbal or kinaesthetic imagery. Children with this type of EEG are therefore particularly handicapped by a hearing-loss and are likely to benefit most from specifically auditory education. Contrariwise, children with normal records but no alpha rhythms are likely to be capable of vivid visual imagery and if deaf, may be able to employ visual aids such as lip reading quite easily. Unfortunately these distinctions are difficult to establish before the age of seven to eight, by which time habits of communication are usually well established, but efforts to extend the analysis to the lower age groups might be rewarding. There is some evidence that these variations in intrinsic EEG rhythm pattern (apart from diagnostic anomalies) are congenital and possibly genetically determined to some extent. This would suggest the existence of inborn traits likely to influence the effect of hearing deficit on the child's personality and development, with corresponding personal needs for special education and exercise.

The effects on the resting EEG of auditory stimuli are very varied, as described by Derbyshire. They can be recognized by careful inspection, but some form of automatic computation can greatly ease the observer's task, particularly when a variety of stimuli are used as Derbyshire rightly recommends. The study of tactile and vibration sense modalities is a valuable complement to auditory analysis since the sense of hearing is essentially a specialized form of vibration sense, with a specific scope and limitation.

The responses evoked during sleep have already been mentioned and in the baby these are the easiest to detect. Such responses are generally referred to as K-complexes following the original work of Loomis, Harvey, Hobart and Davis from 1936 through 1939. Their relation to the rather similar responses evoked in the waking brain has not been satisfactorily analyzed but they have many properties of 'highest level' responses. For example, Pampiglione has demonstrated that if clicks are presented at regular intervals of about 10 sec to a sleeping subject and one or two clicks are omitted, a K-complex often occurs when the clicks would have been expected. This time-conditioning effect has not been investigated in a juvenile population but its maturation and relation to hearing deficit would be an interesting approach to 'unconscious" perception. In adults, sounds which evoke K complexes are often incorporated into dreams with sleep preserving features.

*Cortical Evoked Responses (CER)*

Research into the electric responses evoked in the human brain by sensory stimuli is now becoming one of the major scientific industries. Certainly this topic is responsible for more expenditure on equipment than all the rest of brain research put together. Until the advent of the various types of average response computer such studies were merely qualitative, but now anyone who can buy a miniature computer can measure responses as

accurately as he wishes The most popular type of computer costs about $10,000 and the more versatile instruments about four times as much On the other hand, special-purpose analog devices can be much cheaper and the instrument described by Scott and Weiss is estimated at only $2300 Interestingly enough this latter device resembles in many ways the first average response indicator described by Dawson (in 1951) and the credit for first suggesting this method should certainly go to him

In considering the observations made with the aid of "averagers" we should recall that implicit in every instrument is an assumption and that no one should use a scientific instrument unless he is aware of what assumption he is implicitly making The assumption in every averager is that the arithmetic mean of a series of measurements contains information which is true and relevant with respect to the individual observations When two averages are compared, the significance of the difference between them cannot strictly be estimated without knowledge of the individual *measurements, and this information is irretrievably lost in most averages* For this reason experimenters who must, or choose to, use an averager would be well advised to preserve and inspect the primary records from which the averages were extracted so that they may have some idea at least of the validity of their inferences When reports depend on the average of very large numbers of samples (say N equals more than 50), one can be fairly sure that the experimenter has not inspected the individual responses and cannot therefore know the variance within his population The signal noise gain rises as the square root of N when the "noise" is "white" within the frequency band considered, so that the improvement in resolution over the primary record is most marked with small values of $N$, when the individual responses can at least be appraised qualitatively There is a great need for an averager which would indicate the variance, amplitude histogram or similar measure of scatter at the same time as it displays the mean

### *The Polarity of CER Records*

The general features of the Evoked Response in Man are well documented in the volume of that title published by the New York Academy of Sciences (1964) The least satisfactory feature of this admirable symposium is that *no two workers used both the same* technique and the same experimental conditions The technical discrepancy and diversity in this field must surely be unique in the history of Science, never in the history of human progress have so many paid so little attention to one another The most irritating and confusing discrepancy is the simplest one of polarity convention The traditional physiologic representation of the electric sign of nervous action, the action potential of nerve or muscle, requires that the electro-negativity of the active region be represented by an upward deflection of the indicator Conversely, physicists and electrical engineers decided long ago that the electropositivity should be indicated *above* the baseline

This was entirely arbitrary, and when the electron with its negative charge was discovered by Thomson the convention was regretted but could not be reversed The opposition of these two conventions is seen with terrifying clarity in the publications on evoked potentials Some admit that they do not know, and cannot retrospectively discover, which way is up In the case of alternating, rhythmic waveforms this would be of little or no consequence, but the identification and the interpretation of an evoked potential waveform—a complex transient—depends entirely on its polarity At the present time there is no universal convention, and until one is agreed on and universally adopted in all branches of electrophysiology confusion will be cumulatively compounded

*CER Audiometry*

In relation to the studies of deafness, in the present state of the art, an empirical and pragmatic approach is justified, and there is no doubt that, whatever the physical or physiologic conventions and hypotheses, the responses evoked by various sounds are a powerful and surprisingly accurate method of assessing the sense of hearing at all ages Appleby and Scott have demonstrated responses of classical form in over 160 neonates to tone pips from 600 c s to 4800 c s at intensities down to 40 dB with little habituation up to 200 trials The waveform of these responses is gratifyingly similar to that found by Derbyshire, and is indubitably cortical in origin The latency of the first peak lies at about 120 msec and of the second at 250 msec Much earlier components have been reported but these are almost certainly muscle action potentials, and Bickford (1964) has made a special study of these effects describing this as "centrifugal averaging Their peripheral origin does not diminish the interest of these early responses since they are a part of the basic startle and orienting responses and averaging techniques reveal these components at levels well below clinical visibility In adults the latency of the muscular responses can be as low as 5 msec, indicating a very short reflex pathway, and the possibility of vestibular rather than cochlear participation must always be borne in mind Whenever components with latencies less than 25 msec are observed it is necessary to prove that they are *not* myogenic, and this is often difficult since the reflex tends to follow the same rules as the true evoked responses It would be convenient to have a special term for this reflex and since it is evoked mainly by loud noises rather than tones or speech sounds I have suggested *Psopho*motor Reflex from the Greek word meaning a clash as of shields against one another In Bickford's experiments the effect was best seen in response to clicks alone above 90 dB when the head was strained forward, and we have observed it only occasionally with supine subjects in states of considerable muscular tension The psophomotor reflex sometimes tends to be cumulative in our experiments—a loud click [illegible] kes [illegible] time increases tension anxiety which

augments the reflex and so on. This effect could be confusing if the reflex were used in quantitative studies of hearing.

*Properties of Cortical Evoked Responses*

Comparison of auditory and visual evoked responses is always rewarding and indeed to examine a single modality in a clinical referral may almost be considered negligent. There is one very simple but often ignored difference between the two modalities, there is no indication on the scalp of a true primary specific auditory response. The reason for this is quite simple: the auditory projection area in the superior temporal region is too small to influence the scalp potential field appreciably. In fact, the primary specific visual responses are very small and contribute little to the so-called visual evoked responses which arise mainly in the secondary and tertiary visual association areas. With epidural electrodes over the temporal cortex in human patients Morrell and Morrell (1964) have recorded the primary auditory components quite clearly and have been able to compare them with the more diffuse responses in non specific cortex. The primary components are strictly localized in the expected region whereas those on the scalp extend toward the vertex in a rather peculiar distribution. The Morrells have also studied the geometry of the auditory evoked potentials in premature and full term babies and find that in neonates the distribution over the scalp is very uniform in time and space, as though there were a central generator in a homogeneous volume conductor. In older babies the potential fields become more complex up to the age of about two years, suggesting greater participation of active structures with physiologic propagation. This finding emphasizes the value of the multi-channel analysis, since persistence of the infantile distribution might be an early sign of retarded cortical development.

The changes in evoked responses up to the age of two or three accord quite well with what is known of cortical maturation (Conel, 1939 1947) but the absence of the primary auditory response in scalp recording is a serious drawback. Neurosurgical procedures are now so safe and simple that it might be worth considering the insertion of epidural or subdural electrode strips in cases where identification of a primary evoked response was crucial to the diagnosis or management of an infant patient suspected of hearing deficit.

The difficulty lies in the functional relations between specific and non specific responses, a relation which must determine the basic properties of the brain. Since all scalp auditory responses are from non specific cortex, the information they convey or reflect is contingent on other events and may bear little relation to the strictly auditory or acoustic information. These are essentially "on-effects" appearing at the onset of an auditory stimulus whatever its nature. They signal "To all whom it may concern, something has happened in the ear". There is also an "off-effect" with a peak latency (in the adult) of about 160 msec, compared with the on-

latency of about 100 msec. The off effect may be as large as the on effect and may convey just as much information. The responses to clicks can often (but not always) be resolved into the algebraic sum of the on and off effects and the responses to tone pips usually contain both components also in varying degrees depending on the rate of change in intensity.

*The Contingent Negative Variation (CNV)*

The responses in non specific cortex may be considered as differentiating the auditory stimulus in a rough mathematical sense, that is they are responses to change rather than steady states, as though there were a coupling with a fairly short time constant between the ear and the non specific cortex. The identification and localization of this coupling is a fascinating problem for the future, as is the estimation of the effective time constant. At what rate of intensity change does a non specific response appear? What is the relation between absolute intensity, rate of change and response amplitude? Is the relation the same for rising and falling intensities? Such questions may seem academic but they are extremely practical and urgent because one of the most important developments of these techniques will be toward the study of responses evoked by verbal and semantic rather than physical stimuli. We have already shown that events in which no mean energy change occurs can evoke brain responses if they are subjectively significant. These responses are not the sharp waves of the typical non specific response but quite slow electric tides so to say, that sweep over frontal cortex in the wake of a conditionally significant event and prime the effector regions for synchronous and economical action. I have described these as Contingent Negative Variations (CNV) or Expectancy Waves because of their intimate relation to the statistical information in the stimuli and the attitude of the subject to the situation. Their importance in the study of hearing is that they can be evoked as readily by a word as by a click or tone and indeed a barely audible *significant* word is far more effective than a deafening click.

The discovery of the CNV opens up the whole domain of psychophysiologic study so that we may hope to arrive at quantitative estimates not merely of hearing but of understanding, even in children who are incapable of full cooperation. We have not attempted a systematic study of this problem, which is far beyond our resources, but we have completed a pilot survey of some 50 children with various types and degrees of communication problems with age matched controls and have found a remarkable coincidence between the degree and nature of the disorder and the features of the CNV. In children below the age of four to five the CNV is exquisitely sensitive to social reinforcement and it would seem essential to employ some method like the peepshow technique in order to obtain a satisfactory situation. The importance of parent participation emphasized by [illegible] 1961 [illegible] conditioning is clearly reflected

in the development of the CNV, and this is fully exploited in the peepshow system

The theme which underlies these developments can be described in esoteric terms as an application of information theory, but this description need not be taken too solemnly The simplest illustration is the empirical fact, reported by Davis, Derbyshire, Barnet, and all those engaged in such work, that the repetition rate of brief stimuli is inversely related to the amplitude of the non specific responses they evoke No one seems to have calculated this relation very carefully, but the simplest explanation would seem to be that the more frequently an event occurs, the more probable and predictable each successive event becomes and therefore the less information it contains Here again a practical application suggests itself Suppose that in a particular child the average evoked responses to say 20 *monotonous* auditory stimuli are found to be reduced to one half maximum at a repetition rate of, say, one in 5 sec What will be the effect of averaging the same number of responses to 20 stimuli of the same intensity but *all of which are qualitatively different*? In our experiments this has a dramatic effect in visual presentations, but we have not explored the auditory modality which is technically a little more difficult

## *Conclusion*

In conclusion we should consider what new definitive tests have been proved of value and what outstanding problems remain for future investigation Probably the most satisfactory development has been the demonstration by Davis and his colleagues in St Louis that straightforward measurement of the average evoked response in children can provide data for the construction of audiometric curves at least as reliably as subjective methods These observations have already reached full statistical validity and several interesting features have emerged, not least of which is the lesser correspondence in normal young adults whose subjective reports seem more sensitive than their evoked responses This should provide a clue to the maturational factors in higher nervous activity Among future problems the most exciting possibility is the extension of electrophysiological procedures to the study of semantic transactions in the field of hearing Inevitably this implies the acceptance of social factors as part of the physiological universe, but it would be literally idiotic to consider speech as a function of a single individual in isolation

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# V. MANAGEMENT

## A. Definitions

Chairman Now we will turn over a new leaf and start to discuss the management of the very young deaf child The first question we have to consider is what do we mean by a "deaf child" as opposed to a "hard of-hearing child" and next, "How young is very young?" Here I am taking for granted that we are looking at the pre-school child, unless otherwise specified I will ask Dr Silverman to open the discussion with some general remarks related to some of these questions and definitions

Silverman Mr Chairman, may I, as a useful transition, go back to some of the things that have been said during the last day and a half, because I believe they are relevant to the matter of definition We seem to have been wandering about, in various levels of complexity, among the reasons for doing tests of hearing and the kinds of information that we wish to express about the child

*Objectives and Dimensions* We have spoken about the kind of information that deals with the anatomy of the child The phrase "brain injury" was used when someone said, "His problem is brain injury" He was talking in terms of the child's structure, and I suppose that neurochemical ab normalities would come in the same category

A second class of information deals with the causes of deafness This is etiological information, and we have discussed it at considerable length

A third class of information deals with the behavior of children, whether it be motor behavior or linguistic behavior

A fourth class of information, which has not been mentioned much so far, is what I will term response to therapy Here one describes a child in terms of the way in which he has learned, assuming of course that he has had an opportunity to learn

It will be useful in our thinking if we can find and agree upon one or more such organizing principles I think that some of the differences of opinion that seem to arise among us grow out of the difficulties of making a transition from one category of information to another I suggest that the most useful organizing principle is the one which relates to purposes in seeking definitions

Of course one can avoid the problem of definition by saying that each child is an individual and that we will treat him as such, and forget the

whole problem But this evades the issues to a great extent and does not help those of us who do have need of categories Some of us need to make administrative plans, we need to make suggestions as to how the children should be managed, and therefore we need to find useful ways of describing our children

### *The Audiogram*

One can distinguish among children on a physiological basis, in terms of the audiogram or some related audiometric measure Perhaps it is speech audiometry, perhaps it is pure tones, perhaps we use clicks or pips, but we develop some kind of scale which enables us to classify the child We can then say that a child who had a certain amount of hearing loss when we measured his hearing is "profoundly deaf" or "totally deaf", etc. This is one dimension, decibels, along which children can be ordered from being "normal" to "deaf"

### *Form of Treatment*

Another dimension relates to what you would do for the child One could say that this is the kind of child that needs such and such attention This other child needs another kind of attention Here we might develop an ordered description going from "not too handicapping for the child" to "a great deal of special attention", particularly for children with multiple handicaps or who do not learn readily by conventional methods

### *Mode of Communication*

Still another although rather gross, way of classifying children is according to his mode of communication For some children the primary mode of communication will be auditory, supplemented by vision For another group of children the primary mode will be visual, supplemented by auditory Between these two extremes there will be a rather large "gray" area How these intermediate children are classified is apt to depend on the age at which they were first recognized because the primary mode of the child's communication is affected by the time of onset of his impairment, the time at which it is recognized, his learning experiences, and so on Nevertheless we do know that this gross kind of separation is extremely important for educational purposes and it deserves a good deal of discussion

In summary, I suggest that when we classify children we state explicitly the dimension according to which we are classifying and that we develop some operational descriptions particularly in terms of the objectives toward which we are moving The operational descriptions should deal particularly [illegible] f communication that is appropriate for the child and [illegible]

gn

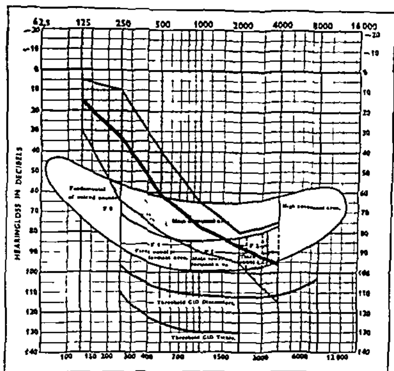



FIG V 1 Group 1 *a* The shaded section represents the composite audiometric curve in group 1 *a* The upper and lower limits defining the area are the group maxima and minima The dark curve in the shaded area represents the mean audiogram constructed on the basis of all the cases in the group (Br )

WEDENBERG At the Conference of Executives of American Schools for the Deaf *in 1937 the following definitions were recommended*

*Deaf* Those in whom the sense of hearing is nonfunctional for the ordinary purposes of life

*Hard of Hearing* Those in whom the sense of hearing although defective, is functional with or without hearing aid

Thanks to the great progress that has been made in amplifier technique the borderline between these two groups has been moved so that many that would have been classed as deaf according to these definitions are now considered to be hard of hearing Defined on a purely physiologic basis, a totally deaf person is one whose sense of hearing does not function in audiometric examinations or lies above the threshold of vibration A hard of hearing person is defined as one with a defective sense of hearing who perceives sound with his hearing organs, however slight that perception may be

We need a classification into sub groups One that I have made and which I hear has been applied is the following

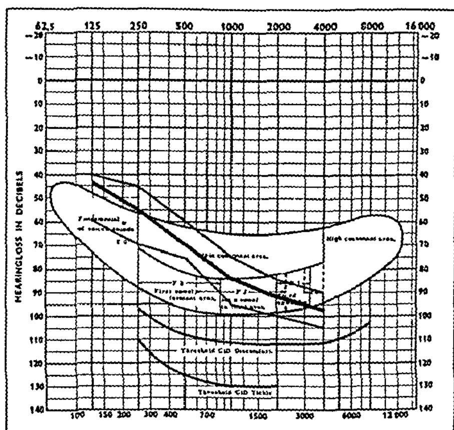



Fig X 2 Group 1 *b* The shaded section represents the composite audiometric curve in group 1 *b* The upper and lower limits defining the area are the group maxima and minima The dark curve in the shaded area represents the mean audiogram constructed on the basis of all the cases in the group (Br)

1 *a* High frequency loss with retained hearing for the low frequencies (Fig 1)
1 *b* High frequency loss with poor hearing for the low frequencies (Fig 2)

2 *a* Flat hearing loss in the formant areas (Fig 3)
2 *b* Excessive flat hearing loss (Fig 4)

3 Vibration sense in the lower frequencies, rising hearing for the higher frequencies (Fig 5)

These groups are clearly differentiated in several respects

1 There are different degrees of difficulty in hearing training 1 *a*, 2 *a*, and 3 are fairly easy to train, whereas 1 *b* and, particularly, 2 *b* are difficult

2 There is a great difference between the ability of the children to use hearing aids Groups 1 *a* and 1 *b* have only little advantage from a conventional hearing aid but a transposer aid is of greater value Group 2 *a* has great advantage from using a hearing aid Group 2 *b* has little if any advantage from any forms of hearing aid Group 3 benefits considerably from an aid

*Acta oto laryng 206*

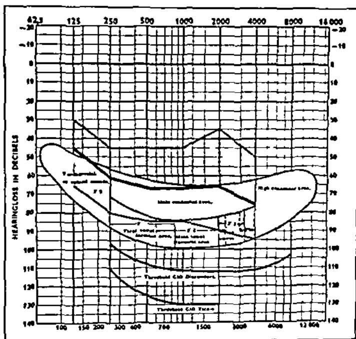



FIG X 3 Group 2 *a* The shaded section represents the composite audiometric curve in group 2 *a* The upper and lower limits defining the area are the group maxima and minima The dark curve in the shaded area represents the mean audiogram constructed on the basis of all the cases in the group (Br )

3 There are large differences in the results of auditory hearing

Groups 1 *a* and 2 *a* are good subjects for auditory hearing They can be trained to speak with normal voices and good accents

Group 1 *b* speak with faulty melodic accents

Group 2 *b* are the most difficult to treat The children speak in a flat voice with defective accents and extremely defective articulation

RØJSKJAER We are running into some difficulties of nomenclature I myself am allergic to the word "deaf", just as Dr Hardy is allergic to the words "pure tone" The word "deaf" and the corresponding word in other languages is, in my opinion, rather misused, particularly in the English-American literature There has been a tendency, up to the last few years, to use this term for all cases of hearing defect from the slightest to the most severe In the latest edition of Davis and Silverman's book "Hearing and Deafness" some opposition to its use emerged The word "deaf", in my opinion, suggests something *negative* something hopeless, and it is a relic of the past, a relic of the time before the audiological era

In Denmark children are called deaf if their speech, due to reduced or extinct hearing and in spite of full and effective use of amplification, does not develop naturally and in due time to such an extent that it is understandable and usable All other children with hearing defects which

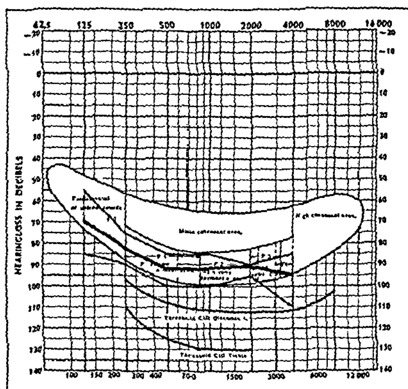



FIG V 4 Group 2*b* The shaded section represents the composite audiometric curve in group 2*b* The upper and lower limits defining the area are the group maxima and minima. The dark curve in the shaded area represents the *mean audiogram* constructed on the basis of all the cases in the group (Br )

are severe enough to produce a handicap are called "hard of hearing" We have a better and easier word in Danish than the English "hard of hearing", and the condition may be characterized as severe, moderate, or slight I should like to confine the term "deaf" to patients who are completely devoid of hearing, i e "totally deaf" We could call the other patients "profoundly hard of hearing", but it might be better if we could find an entirely new word for this group who have some hearing but not enough to develop language and speech primarily through hearing even with the use of amplifying apparatus I hope that other participants will make some suggestions, I have no solutions myself

I think we need further classification which might go something as follows

*Group 1* The totally deaf These persons we may properly call deaf

*Group 2* The group with a new name which we might subdivide into

*Subgroup A* Children with a small residue of hearing which is not practically usable with the hearing aids available today

*Subgroup B* The children with a residue that is large enough to be usable with present hearing aids

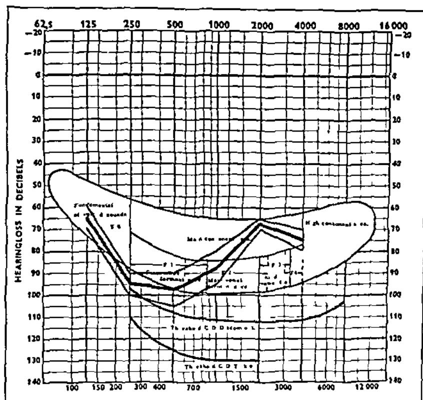



FIG V 5 Group 3 The shaded section represents the composite audiometric curve in group 3 The upper and lower limits defining the area are the group maxima and minima The dark curve in the shaded area represents the mean audiogram constructed on the basis of all the cases in the group (Br)

*Group 3* Several hard of hearing children These children need specially educated teachers to work in special schools or in specially equipped classes A small subgroup of these may be taught within the framework of schools for the normal The speech of group 3 children will develop mainly on the basis of hearing but it will have defects and require correction by trained teachers

*Group 4* Moderately hard of hearing children who can be educated in normal schools and classes with only occasional special classes The children may use hearing aids or perhaps some special equipment such as induction or frequency modulated transmitting systems They still need considerable special educational help from *specially trained teachers*

*Group 5* Slightly hard of hearing children who get their education without difficulty and in normal classes and rarely may not require hearing aids This is the last group

This grouping system does not include children who become deafened later in life after having acquired language and speech in the normal way

CHAIRMAN. This is a very helpful classification. It is helpful even without all of the words or special names because it represents a graded series of *difficulties, a series of handicaps.*

BRILL. I disagree in part with what Dr Røjskjaer has just said because it seems to me that his operational description of definition jumps a step. It is, *as I understand it, based on how speech is used rather than on the* extent to which hearing is used. To illustrate, think of the boy who, at eight or ten or twelve years of age, loses his hearing from meningitis. His hearing loss, as measured by audiometers, is as severe as it can be and yet his speech is virtually normal or can be so if it is properly maintained. To me it is not appropriate to classify this individual as hard-of-hearing simply because he has virtually normal speech. This does not fit our particular *educational problem.*

I don't object to using the word "deaf." I use it to describe the individual who cannot hear and understand *connected speech.* The simple test is, can this person with eyes closed understand a sentence that lies within *the range of what one may reasonably expect in terms of his age and* experience. Perhaps he can hear a few sounds or even some individual words, but for me this individual is deaf if he does not get the total concept of the sentence. If, on the other hand, he can understand the connected phrase with his eyes closed but he has a hearing loss, I classify him as hard of hearing.

DAVIS. *Dr Brill, do I infer from this that according to your definition* it would be possible to educate a person out of his deafness?

BRILL. Occasionally. By education we might move the child across the borderline from the group of those who do not understand connected speech to the group that does. I think it is obvious that the degree of handicap can be modified by education and that is why we try so hard to educate *these children.*

Let me illustrate further in terms of the classification of children in our school, the California School for the Deaf. Partly because of the pressures of long waiting lists and also because of the other opportunities *for education* in the State of California, we try very hard not to accept any children who would be classified as merely hard of hearing in the sense of the definition that I just gave. We don't think that we have any, particularly among the younger children, but we might be wrong *once in a* while. Among the older children we have a group of 14 whom we call hard of hearing. For the most part they are children who have been in other kinds of programs and have *not been successful and so they have come to us.*

We have another group of 16 children whom we call "trained hard of [illegible]." When a naive person talks to a trained hard of-hearing child his [illegible] is that he differs very little from the other group *of hard of*

hearing, but those who know them better realize that there is a difference in that the trained hard of-hearing child is able to operate much more freely in the realm of language and the understanding of the speech that he has been taught If you use vocabulary that is likely to be unfamiliar to this group they don't get it through the ear alone This is in contrast to the other hard of hearing

For the 14 hard of hearing children the average decibel loss at the three frequencies of 500, 1000 and 2000 c s is 58 dB That is the mean for the group For the trained group as a whole the mean is 74 dB (ASA)

*(The following is a condensation of comments submitted by Dr Brill following the Conference)*

The children in the lower school at the California School for the Deaf at Riverside and some of the children in the Elementary school were evaluated by the supervising teachers of their respective departments in terms of the apparent usefulness of their residual hearing with amplification The children actually divided into almost equal thirds among three categories, those who benefit greatly from amplification those who benefit to a slight degree, and those who do not benefit from it at all

All of these children are deaf, not merely hard of hearing Those who fall in the category of benefiting a great deal are able to discriminate sounds to the extent that when there is a choice of distinguishing among two or three words that are known to the child or when the number of syllables within the words is varied the child is able to select the correct word This benefit they had achieved through auditory training These children are able to make enough fine discriminations in sound to be of tremendous value to them in learning to speak clearly and intelligibly and to some degree in assisting them to understand the speech of others

The children in the second category, who benefit to a slight degree, can distinguish between gross sounds The child may be able to distinguish the difference between the sound of a bell and the sound of a horn or may be able to indicate whether there was one drumbeat or two drumbeats or perhaps three

The children in the third category, who do not benefit at all from the hearing aid, are not able even to distinguish between gross sounds although they may perhaps respond to the extent that they can indicate whether there was sound present or not as in an audiometric test

The audiometric hearing levels of these children were determined and the children were distributed, by categories, according to the range of frequencies to which they gave a positive response The average hearing level for the prime speech frequencies was also calculated, where possible, but the numerical results are difficult to assess because in many cases there was no response at the limit of the audiometer and therefore the average is indeterminate Examination of these distributions, which were shown as lantern slides at the conference, reveals an interesting relation

For these children, the range of frequencies that are heard is a better predictor of the degree of benefit received from amplification than the average of the decibel loss for the three prime speech frequencies Eighty-five percent of the children who benefit greatly from amplification respond to the 2000 cps tone, over 60% to 4000, and 43% to 8000 By contrast a much smaller percentage (32%) of those who benefit only slightly can hear 4000 cps At the other end of the scale, 42% of those who did not benefit at all gave no response above 500 cps Nine of these children did not even have "residual hearing" and failed to respond to any audiometric tone

A group of elementary school children are assigned to a class in which finger spelling is used simultaneously with speech because these children, although they had been given the opportunity of being taught exclusively through oral means of communication, did not seem to be able to benefit particularly from it In this group of 28 children there are 14 (50%) who receive no benefit from their hearing, even with amplification, 8 children (28%) benefit to a slight degree, and 6 children (21%) benefit a great deal from their hearing The 6 children who do benefit a great deal reap this benefit in the improvement of their own speech, but their hearing is of no particular benefit to them in the receptive process Here again, 4 of the 6 children who benefit a great deal can hear both 4000 and 8000 cps while only two children in each of the two other (larger) groups can hear 4000 and none can hear 8000 cps Ten of the 14 children who receive no benefit from their hearing do not respond to any frequency above 500 cps Actually, in this entire class of 28 children there are only two whose average decibel loss for the prime speech frequencies is less than 80 dB (ASA) They must not be confused with the hard of hearing or the trained hard-of hearing children who were mentioned earlier in the conference discussion

Finally we evaluated an elementary school class of 8 children who had been designated as "aphasic" Without going into detail, it is perfectly clear that with one possible exception these children also are probably truly deaf, not merely hard of hearing

### *Audiometric Zero Reference Level*

DAVIS I suppose you are using the American Standard reference level for your decibels Perhaps this is the time, while we are speaking of an average hearing loss expressed in decibels, to call attention to the reference levels of our audiometers, which differ on the two sides of the Atlantic I am sure we are all familiar with this and do not need to discuss it We will simply place it on the record that audiometric readings, whether they are individual readings or average hearing losses, mean something about 10 decibels different by the American Standard from what they do by the h Standard or by the new International Standard

SILVERMAN There is a vast amount of literature in which this situation is unfortunately not called to the attention of the reader I am interested not only in the speedy and universal adoption of the International Standard but also in dealing with the literature that ignores these differences

DAVIS Well, as with the polarity of our electroencephalographic records, if we cannot be uniform at least we can be explicit The designation "ASA" can be used to mean the current American Standard established in 1951 "ISO" means the International Standard Organization s recommendation of 1964 It will be absolutely essential from now on to designate clearly on all audiograms or wherever the reading of an audiometer in decibels is mentioned, which standard is being used

W HARDY Even in this meeting there have already been shifts back and forth in reference levels I would strongly urge, in addition to this very positive suggestion for the International Standard audiometric reference level, that when we write our reports or submit our data to the public we take care to specify whether our measurements are sound pressure levels or sensation levels or hearing levels This is rarely done with sufficient care

While we are talking about hearing losses, where that term is adequate, I would like to suggest further that we talk about 'sensitivity levels" rather than "acuity levels" This is a source of utter confusion to a great many people who are not quite up-to-date with modern terms

DAVIS In regard to audiograms, I propose that in the publication that will result from this conference, we standardize on the "ISO-1964" Reference Zero level This in principle has already been accepted by three of the major otological and audiological societies in the United States, namely the American Otological Society, the so-called "Triological Society" (American Laryngological, Rhinological and Otological Society, Inc ), and the American Speech and Hearing Association Any audiograms accepted for publication in the journals of any of these three societies after the 1st of January 1965 will have to be plotted on the ISO 1964 scale I believe that we, as an international group, should certainly do likewise I shall take this for granted in editing our proceedings

WALTER Would you repeat which way the 10 decibel correction should be applied?

DAVIS The ISO standard is more sensitive, that is, you reach audiometric zero at a lower sound pressure level Therefore, you add 10 dB roughly, in order to translate from ASA to ISO hearing levels The 10 dB is a round number the adjustment ranges from 15 dB to 6 5 dB depending on the particular frequency, but 10 dB is a good round number for the

frequencies most important for speech. We Americans must learn to shift our mental criteria for classifications, for what is a handicap, and so on. It is much more than just recalibrating our audiometers.

## *Classification of Children*

LOWELL: I would like to hark back to Dr Silverman's point about operational definitions and take brief exception to Dr Røjskjær's classification system. If our criterion, as I understand it, is to depend upon where the children are in school, I think that this may not be an appropriate classification because different communities have different kinds of educational opportunities. A child might be in the third class simply because there are no fourth or fifth class opportunities available to him, or vice versa. I therefore think that definitions in terms of school placements leave something to be desired.

CHAIRMAN: Yes, now we are fundamentally considering the degree of the child's present handicap as opposed to his physiological and mental potentialities. These latter may be something quite different, and the child's status is subject to modification.

A. EWING: My thought is that if we attach labels to children we must be prepared to change them during the child's education, whether before admission to school or later.

CHAIRMAN: Dr Huizing, do you have something to add to the discussion of definitions?

HUIZING: The problem of definitions is very difficult, but it is nevertheless very important that we come to some agreement as to meanings of our terms and also recognize, as Dr Silverman has pointed out, that we may require different sets of definitions for different purposes. Let me illustrate the problem.

In my country (Netherlands) we have two different types of education in two different types of school. We talk about schools for the hard of hearing and schools for deaf children. Sometimes it happens that, as the result of effective training and education, we can transfer a child from the school for the deaf to the school for the hard of hearing. This means that in such cases it might be very difficult to label a child in two classifications or to change his classification.

The classification might be based on the audiogram as a measurement of impairment, but it might require partly an estimate of the child's intelligence, and this is difficult to put into figures. A point which is closely [illegible] this is of course the education of the child, and therefore I would [illegible] this problem of classification as an introduction to a discussion

of the use of amplified sound, because we now have many examples of the assistance rendered by amplification to hearing-handicapped children

In my country we started to use amplification for the hard-of hearing child in 1947, just as a kind of experiment The results were so favorable that we soon had to apply amplification on a much larger scale This led to a new start in 1950 when a group of children three years of age got hearing aids They were selected because they had some good residual hearing and good intelligence This isolated group reacted excellently and soon thereafter all pupils at school for the hard of hearing had to be fitted with hearing aids, and in turn in 1955 hearing aids were introduced into home training

In home training the child is taught by the parents under the guidance of the audiology institute at a school for the deaf The results have varied widely, sometimes they are very good and in other cases and social circumstances it is impossible to get even reasonable results

SILVERMAN I think it is implied in what I suggested that we should continue to consider the possibility of reclassifying the child If gradations of ability to communicate determine classification, for example, we may educate him to better communication I think that it is very important that the classification should not be static

WHETNALL At least it could be stated, when a classification is made, whether it is before any training has started or subsequently Perhaps the child starts to learn to hear It is the child s ultimate ability to overcome its handicap which really matters

TERVOORT I think that a useful distinction is one that Fr Van Uden has introduced in our school It is a very handy operational distinction The *deaf* child is defined as "a visual being for whom communication comes mainly through the eye with eventual help from the auditory system" The *half-hearing* child is "an auditory being for whom information comes mainly through the ear with the eventual help of the eye" This is a distinction that usually works when you have to decide whether the child, in the long run, is a child for the school for the hard of hearing or for the school for the deaf

WALTER I think that Dr Silverman's suggestion about modality preference is both very useful and very powerful, because there is some evidence of inborn modality preferences in children which are difficult to assess but which seem to exist This might throw considerable light on a difficulty which we all have, both with relation to blindness and to deafness Those who are born, for example, with an "inbuilt preference" for auditory imagery are particularly handicapped if they become hard of

hearing, whereas those born with a preference for visual imagery do a lot better Of course the reverse applies to blindness I think that this could be considered as a sort of sub-classification in the scheme

## B. Differential Diagnosis (continued)

Chairman On the agenda we find the question, "What important medical, psychiatric, or psychological *conditions, including congenital* aphasia, may be confused with peripheral (inner ear) deafness?" Some problems of differential diagnosis and tests appropriate for it were considered yesterday, but I believe that several members have more that they would like to add on the subject

W Hardy. I think that what Dr Silverman has just expressed is a very wise warning We seem to be afflicted with a patient load of young preschool children, who simply do not come in nice, neat packages describable in terms of hearing loss We do see some simple textbook cases and it is a pleasure to work with them, particularly if you see as many as three or four consecutively For the most part we are forced, clinically, to pay attention to which sensory modalities are working, how well they are working, which ones are not working, and what, if anything, can be done *in the way of direct physiological remedy* When you try first to understand then to select, then help to enhance the function of residual modalities, with the fond hope that some of the affected ones may improve as the child learns, you find yourself involved in all the details of the high risk group that Dr Janet Hardy outlined yesterday. Often, with a two-year-old child, one is hard put to it to distinguish between developmental defects and the effects of various kinds of trauma Then there is the total effect of the combination in relation to the child's history, his behavior and his expectancies Our group hasn't yet found out very much about another extremely important aspect that has already been mentioned, namely the total effect of what you might call "psychosocial deprivations" This is the reflection in the child of the aggregate of his environmental experience I am firmly convinced that all of these things have to be considered before one can presume to make a diagnostic judgment, let alone a prediction

It seems to me, Mr Chairman, that everything that's listed here, medical, psychiatric, psychological and congenital factors, may each and all, at some time in the early period of the child's life, be confused with the old-fashioned peripheral hearing loss This has been demonstrated repeatedly with many sub-normal children, with *obviously brain-injured* children, and with many so-called psychotic children I know of no way to deal with this except to find out what is working and what isn't working, to describe in detail as carefully as I can, and then to try to get the child

started as soon as possible on a sequence that we have some years for lack of a better term, been calling 'diagnostic teaching"

SILVERMAN· I would like to emphasize a point about "psychosocial factors" I too have come to have a great concern about our views of what ultimately will or should happen to a child My concern revolves about the handicapped person whom we have, in a sense relegated to a marginal existence because we think that this is "the normal thing' Many of our predictions are made only on the basis of how well a child communicates, but there is also his relation to his total environment which is exceedingly important My concern grows out of talking with a good many adults who would be judged to be close to normal by audiometric tests at least according to their aided thresholds using their hearing aids and even according to the way in which they communicate, but who experience a good deal of psychic distress by being in a situation that is constantly marginal and from which they have no relief They find it difficult to move about in a world that is gray for them And sometimes this process starts right at the beginning, in the management of a child and in some of the predictions and prognoses that we make to parents It starts right at this age and not in adulthood

CHAIRMAN Dr Hawke, yesterday you gave us some very helpful psychiatric comments that are still pertinent Do you have anything more that you would like to add?

HAWKE I would like to add a little about each of the four major syndromes They are based, to some degree, on clinical observations and therefore they obviously have the defects of lack of controls and lack of validation

*Autism* Concerning autism, I think that there is increasing awareness that autism is not an all-or-none phenomenon it is a spectrum Personalities vary from the extrovert to the introvert and I suspect that the distribution of individuals along this scale is on a normal or gaussian curve Within this curve you will find the autistic group We now diagnose it more freely than we used to, on the basis of minimal to moderate abnormalities of behavior

Another point is that behavior patterns change with growth, irrespective, I am sure, of therapy The changes have occurred in children without specific therapy We may see a child who was extremely autistic at two years of age but at four this child was obviously a retarded child without significant autism I think we recognize that there is perhaps what we can call a *primary autism* which relates very often to a schizophrenic process Other factors may increase this, and there are children who are blind who are also autistic and children who are deaf and also autistic

There are a number of deaf children with associated autism which must be considered part of the syndrome

*Familial trends* Concerning the developmental delays and maturation lags that we were discussing, I ought to mention that there is very frequently a strong family history. Sometimes there is a comparable condition in other members of the family but sometimes you will have one child with a language delay and another with a visual reading disability There is also, I am quite certain, a very strong relation to "handedness" At least 75% to 80% will give you a history of left-handedness or ambidexterity within the family The meaning of this is uncertain We do have some idea of the percentage of right-handedness and left-handedness in the community However I do not believe that an adequate survey has been made that includes the marginal group, the ambidextrous individual, the right-handed but left-eyed individual, the right-handed individual who plays sports left-handed, etc This is important because the 75% or 80% that I am talking about may turn out after all to be very close to the normal proportion On the other hand there does seem to be a specific relationship since in these families there is a tendency for these irregularities of development

Dr Silverman and Dr Walter have already commented on another point that I wanted to make, namely the variation of people in respect to their auditory and visual skills and the importance of modifying your techniques depending upon the basic inborn abilities of the child

## C. Medical and Surgical Procedures

Chairman We will now take a quick look at the conditions that can be detected and can be helped by medical or surgical procedures Dr Ireland, you have been strangely silent so far Would you summarize this field for us?

Ireland I have been strangely silent, sir, because I have been amazed at how little I know, but as an otologist, I think we can divide the remediable group to advantage into certain categories I think that our remediable group is the *conductive* group

*Middle Ear Disease* I need only mention wax and external otitis in passing I must say more however, about true middle ear disease I think that the most important cure or remedial treatment for middle ear disease is removal of adenoids, and I am sure that everyone of you who tests children knows the remarkable effect that can follow the removal of adenoids

An important condition is *serous otitis media* I like the term "glue" ear to describe a late stage of this condition Serous otitis media is something that is missed frequently, and we are very much indebted to Dr

Hoople at the University of New York, formerly Syracuse University, for his new ideas about it It is fantastic when you have a patient who doesn't hear and you do a simple operation and he hears immediately You don t need an audiogram or anything else, he just hears and there's a smile on his face The ones that are not immediately relieved can, of course, be treated *by putting in a polyethylene tube (it's a simple matter to put it in)* and leaving it there. Many of these ears are remediable and do subside

We then come to something more difficult, the ordinary *perforation of an eardrum* Now newer operations for the closing of the perforation of an ear may give an improvement of up to 20 or 25 decibels These myringoplasties are well worthwhile In the case of infection that requires mastoidectomy, and in ordinary tympanoplasty, a simple graft is put on the eardrum I am surprised that we otologists were so long in getting this kind of surgery going We have to thank our German confreres very much for their information and demonstrations

*Congenital Malformations* To get to less common conditions you are quite familiar with the congenital ear The *operation for congenital ear* used to be a bit formidable We had no idea where the facial nerve might be located and there might be all sorts of congenital anomalies that were difficult to foresee I think that my other friends in otology will agree that with modern X-ray techniques and with modern methods of approach, the congenital ear is not a formidable operation, and its correction is a very excellent aid to good speech I think that the patient with bilateral congenital ears who has been operated upon is much better off than if he had years of speech training

*Trauma* There is the accident case, which is more common, I think, than we sometimes realize The incudostapedial joint is dislocated This *can be corrected by surgery and normal hearing restored*

*Otosclerosis* I have purposely left otosclerosis to the last, because we think of otosclerosis as a disease of older people But otosclerosis is not necessarily a disease of older people, it can occur in children as well The main thing is to recognize this condition It is remediable and the results of surgical treatment have been excellent

*Sensory-Neural Deafness* When we get to the nerve-deaf patient I don't think there is anything that is really remediable from the surgical point of view I would like to be corrected if I am wrong The only thing to remember is that perhaps in some combined cases we may be able to raise the hearing enough for the patient to wear a hearing aid successfully This is well worthwhile

I won't say anything about *acoustic neuroma* In the treatment of *Menière's syndrome* by using ultrasound we may be able to preserve hearing, that is, if there is not too much cochlear damage

*The audiogram* Now what is the useful part of this to us as surgeons or otologists The useful part is that you audiologists can tell us that a patient has an air bone gap Also we want to know what kind of hearing

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*The audiogram* Now what is the useful part of this to us as surgeons or otologists The useful part is that you audiologists can tell us that a patient has an air-bone gap Also we want to know what kind of hearing

the patients have As Dr. Barr said this morning, we want to compare our post-operative results with our pre-operative results This is what tells us how good the operation was and how well it lasted The pure-tone audiogram is the best measure for peripheral and particularly for conductive hearing loss

McHUGH: I would like to stress the importance of *regular otological care* for all children who are deaf We have all seen, time and time again, children with severe sensory hearing losses who have been subject to recurring conductive losses superimposed on the sensori-neural by inflammations, allergies, and so on This superimposed conductive loss may alter the child's status and category in terms of his education completely for weeks and for months on end, so I feel that every child who is deaf, and regardless of the classification he's in, should be examined from time to time to be certain that the middle ear mechanism is intact

I would like to note for the record recent observations that there is a tremendous difference between the total effect of a conductive hearing loss due to the presence of fluid in the middle ear, i e secretory otitis media when this occurs in a child of school age and when it occurs in an infant under the age of one year In the past year I have been impressed by three infants all under the age of nine months who behaved as if they were profoundly deaf but who, after the removal of chronically infected adenoid tissue from the nasopharynx, had not only the clinical evidence of recovery from the secretory otitis media but who then appeared to be perfectly normal in terms of their auditory behavior after the middle ear function had been restored One cannot fail to be impressed that the level of the conductive hearing loss, i e approximately 30 dB, that occurs with secretory otitis media should create such an apparently severe sensory deficit This would suggest that untreated, persistent middle ear disease due to inflammation or allergy, or both might well result in a degree of sensory deprivation and subsequent behavioral changes in normal development that one would not expect in an older child

I would also like to record a word of caution regarding tympanoplastic or other reconstructive middle ear procedures on young children The literature abounds with enthusiastic reports of success in adults with these various procedures and it would appear to be very easy to repair ear drums or perform various types of modified mastoid operations to "provide freedom from disease and restore their hearing" In my many years of experience at the Montreal Children's Hospital I have been impressed many times by apparently beautiful technical procedures performed on the middle ears of infants and children that a few months later were neither free of suppuration nor successful in restoring the hearing

I would like to repeat that when we examine children carefully we find a tidy number who have conductive hearing losses, 60 to 70 decibels (ISO), due to various types of middle-ear congenital abnormalities They

may have perfectly normal external ears, normal canals, normal drums, but with, for instance, ossified stapedius tendons One patient of 12 years, with perfectly normal malleus and incus and a good footplate had no crural arches at all Also we see a few cases every year of closed head injury producing disarticulation of the ossicles

The differential diagnosis of sensory and or neural hearing loss in children is usually impossible or at best extremely difficult in young children due to our present lack of appropriate tests that may be compared with the more elaborate hearing techniques available for adults Nevertheless I believe we should keep in mind that all sensory neural hearing losses in children are not all untreatable I have seen a number of children who have shown a flat audiogram quite compatible with the diagnosis of sensory hearing loss that might justifiably be called juvenile cochlear hydrops in view of the variations that occur from time to time I believe that cochlear hydrops is an expression of a psychosomatic disorder and as in adults some of these children can be helped by medical treatment with certain anti-depressant drugs

## *Congenital Malformations*

BARR The question is asked whether *congenital malformations* of the middle and/or outer ear are a significant problem We have had some experience with such cases at the Institution of Audiology for Children at Karolinska Hospital in Stockholm They represent only a few percent of the total material of 961 children There are included only cases in which the hearing defects had been so severe that language and spontaneous development of speech had been retarded or did not occur at all We were in contact with most of these children before they were three years old and their case histories are based on conversations with their parents and from medical examinations The causes of deafness as far as they could be identified, are given in Table V 1

Thirty-six children had bilateral congenital atresia and/or malformed ossicles, which means about 4% of the cases for which case histories were available Out of 904 children born before 1959 only 20 had this malformation Among the children born 1959–1962, however, this type of deformity was found in 16 out of 57, and of these, *fourteen of the mothers had taken thalidomide in early pregnancy* Except for the thalidomide period not more than 2% of the deaf seem to have malformations of the middle or outer ear Among the 20 children in whom there was no history of thalidomide medication, other deformities were extremely rare and included three cases of typical Treacher Collins syndrome There were two children with slight facial paralysis and two with total deafness, while the others had conductive hearing loss On the other hand, all the thalidomide children had at least one form of cranial nerve paralysis, and the proportion of total deafness was much higher—8 out of 14

TABLE V 1 *Causes of Deafness*

| | |
|---|---|
| Hereditary | 146 (16%) |
| Maternal rubella | 136 (14%) |
| Congenital atresia and or malformed ossicles | 36 (4%) |
| *Unclassifiable* | 385 *(41%)* |
| Probably congenital | 703 (75%) |
| Anoxia, birth injuries cerebral palsy, prematurity (under 1500 g) kernicterus | 135 |
| Probably acquired perinatal | 135 (14%) |
| Meningitis encephalitis and/or dihydrostreptomycin | 69 |
| Middle ear disease (cleft palate 15) | 23 |
| Mumps | 1 |
| Sudden deafness unclassifiable | 1 |
| Trauma | 1 |
| Acquired postnatal | 95 (10%) |
| | 933 (100%) |
| No case history | 28 |
| Total | 961 |

There are theoretical and practical possibilities of improving the hearing of some of these children with congenital malformations by an operation But especially the operation of *atresia* cases is very difficult and although some cases have had encouraging results, the result is far from satisfactory for the majority The prospects are much better for the cases in which the malformations are confined to the ossicles Many cases of pure conductive hearing loss without atresia will probably be diagnosed as early cases of otosclerosis and never be recognized as congenital malformations (House *et al*, 1958, Sooy, 1960) The prerequisite for operation is a firm diagnosis with a full knowledge of the inner ear function in both ears

The best age for operation depends primarily on how early a hearing test can be carried out with air and bone conduction and reliable masking so that the function of each ear can be tested individually This can hardly be done before the age of three An operation requires long and difficult postoperative care and it is therefore advisable to postpone an operation for a few years until the child is older and easier to handle This is permissible because these hearing defects can very well be compensated with amplified sound and auditory training

A contraindication for an operation is the absence of inner ear function in one or both ears *In the latter case an operation is of course* useless, and in the former the operative risk is too great compared with the probable benefit The absence of mastoid air cells and facial paralysis are not direct

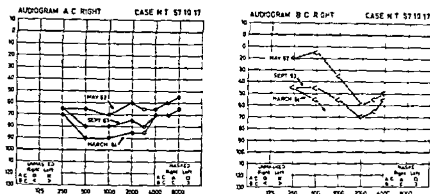



FIG V 6 Pure tone audiograms (Br) from a child with endogenous (hereditary) combined hearing loss in the right ear showing spontaneous deterioration during an observation time of two years Left ear no response Case history does not indicate any middle ear infections and ear canal and tympanic membrane are normal on inspection A surgical exploration in such a case is absolutely contraindicated

contraindications for an operation, but they reduce the chances of a good result

The groups "hereditary" and "unclassifiable" in Table V 1 include a number of children who in addition to a sensori-neural hearing loss have also a conductive hearing loss, indicating a defect in the middle ear Operations have been performed to improve their hearing and promising results have been obtained, but we do not yet have any long-term results (Schlosser *et al*, 1964) In these cases the inner ear is probably abnormal and very sensitive to all operative measures, and when manipulating the stapes the risk of causing a "dead ear" is great Dr Wedenberg and I have reexamined children with sensori-neural hearing loss up to 15 years after the first reliable pure tone audiogram In cases with definite hereditary deafness many of the children show a spontaneous progression of the hearing defect Here even cases with conductive components have been seen that have progressed into nearly total deafness without an operation having been done Operations on such children should only be done in cases that can be followed up continuously and thus give us better knowledge, and of course only if the child hears with both ears

RØJSKJÆR In Odense we have had disturbing experiences with surgical restoration of hearing in congenital malformations of the middle ear and the osseous ear canal, usually we are not prepared to operate these cases if they are unilateral If the defect is located laterally in the outer ear, whether it is in the auricula or the ear canal, we try to correct it surgically, to make the patient hear normally and for cosmetic reasons In cases with malformations all over the external and middle ear and in bilateral cases of that type operations are indicated *to establish anatomic conditions for using an ear insert* in connection with hearing aid treatment It is more

convenient and better for the patient to use an ear insert even if it has no connection with the middle ear rather than using a bone conductor mounted on a head band As you see we are rather conservative partly because of the results obtained and partly due to our hesitation to cause suffering to our patients which they did not have before the operation was carried out i e chronic suppuration a facial palsy vertigo or the like *The Danish population is rather audiologically minded* and therefore we prefer creating conditions for an effective audiological treatment or if the anatomical condition already is present without operation we start an audiological treatment and sustain it for a lifetime rather than undertake some very questionable operations We do operations but we are very critical when we make our indications If we have to operate we usually start the series of operations around the age of six or seven

CHAIRMAN Dr Bertrand do you have a comment on otitis media or the glue ear?

BERTRAND Serous otitis media and the glue ear constitute a serious problem We do not know with absolute certainty the physiopathology responsible for the production of middle ear liquid It might be a transudate coming from a physiologic obstruction of the Eustachian tube or it could *be secreted presumably under a parasympathetic influence* A new hypothesis is that it is an auto immune disease

We have been using a polyethylene tube quite frequently for the last four years in cases of serous otitis media and we believe that in many cases we arrest the evolution of the otitis media before it reaches the second stage We consider that the glue ear is probably a second stage of otitis media which we see in the first stage as serous fluid In the second stage the consistency of the liquid becomes rubbery and adherent to the mucosa and it is almost impossible to remove it completely by a myringotomy and aspiration The disease progresses if not treated to a third stage adhesive *otitis media*

The use of the polyethylene tube has reduced considerably the evolution of serous otitis media to the stage of the glue ear especially in recurrent cases When at the first stage we may sometimes proceed only with a myringotomy if the liquid is limpid but whenever we have a recurrence or even if the fluid is slightly sticky we will routinely use a polyethylene tube

The function (role) of the polyethylene tube is not so important for continued drainage but to establish a normal pressure in the tympanic cavity

In cases of glue ear which persist after a conservative treatment with a polyethylene tube we believe in doing a mastoidectomy to remove the glue and in these cases we will put in a polyethylene tube during the operation to obtain adequate ventilation and pressure in the middle ear

This somewhat resembles the principle described this month October 1964) by Sirala

I would like to ask a question about congenital atresia At what age do you believe a unilateral operation for bilateral congenital atresia should be done?

CHAIRMAN Dr Ireland do you want to answer that last question

IRELAND No because I don t think I can I think that a unilateral congenital atresia probably *shouldn t* be operated on On the other hand Senator Sullivan and his group who were with us last night *do* operate on them

CHAIRMAN Dr Barr do you have an opinion on the age at which operation for congenital atresia should be performed

BARR As I said before you must be quite sure you know the function of the ear and you can t be sure before three or four years

CHAIRMAN In other words you have to wait for tests that really satisfy you so that you feel that you really know what you re dealing with

Well with this we will leave the otological aspects They seem to hinge clearly around conductive impairment and a great deal can be done surgically for any variety of conductive impairment

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3 SCHLOSSER W WINCHESTER, R and GOLDMAN B 1964 Further experiences with the diagnosis and surgical management of congenital mixed deafness *Laryngoscope* 4 773

## D PARENTAL SKILLS AND ATTITUDES INCLUDING HOME TRAINING

CHAIRMAN We will now assume that all that can be done medically and surgically for the deaf child has been done What next? On our agenda we have the topic Improving parental skills and attitudes Lady Ewing will you open the discussion on this topic?

### *How to Help and Guide Parents*

LADY EWING The main subjects of this statement are home training and parent guidance and we are beginning to see the great possibilities of

guidance to parents of deaf and hard of hearing school children, as well as for parents of very young children.

The principles and adaptations of the methods are applicable to parents of deaf children of any age, in teaching their children to talk.

Parental cooperation is established at the outset by explaining and demonstrating the effects of the child's deafness to the parents, particularly in terms of loudness levels at which the child can hear. The child's possibilities are put to the parents and the question is asked, "What do you want for your child?" It is made quite clear to the parents that, whilst explanations and demonstrations can be given to them, they themselves need to do the work of training their child. *If the parents accept our help*, they have also to accept responsibility for attending regularly at both guidance sessions and meetings.

### *Guidance Sessions*

Parents who accept guidance are expected to attend the University Audiology Clinic at least once a fortnight, either for individual guidance sessions or for group meetings. We have found that to give support to the parents, to help them to accept their child's deafness and to face and deal with problems arising from it, is an essential part of our work. Meetings at which parents can take part in informal discussions with one another and see demonstrations by other parents, with their children, contribute to this support.

The subjects which need to be treated in guidance to parents are:

(1) *The use of vision in association with hearing to stimulate children's interest in and knowledge of sources of different forms of sound*, so making them meaningful. We want each child to be given as much experience as possible of sounds, other than those of speech, which are so important in providing information about the everyday world and which serve as reference points in our orientation in that world. Because, as yet, many deaf children are unable to localize sounds with hearing aids, we find that they benefit greatly from training in identification of sources of sound and in making sounds that amplification enables them to hear.

(2) *The use of vision, to supplement use of hearing aids, in understanding speech.*

(*a*) In routine situations normal to the care of children—at meals, bedtime, etc., especially during dressing and undressing.

(*b*) In contrived play situations and in free play and, as soon as a child is old enough, in helping mother about the house, cooking, going shopping, etc. and helping father in the garden and in the garage. *It is not enough for deaf children to know how to do things.* Both for concept formation and for communication, they need skillful training to understand and use the relevant vocabulary and language. They must be able to talk *about people, things and events.*

Play material and activities must be appropriate to cooperative play and, of course, to the age of a child

Parents need help in using methods of drawing a child's attention to the face of a speaker, for instance by holding up toys, by withholding a toy for a moment or withdrawing it by introducing a new toy In situations which are utilized or contrived by parents for home training they need to learn to encourage children to listen attentively to their voices and to watch their faces while they are speaking Toys have to be selected and used in such a way that in these situations children do not become involved in manipulation and solitary play As a result of this policy, a child who has at first been motivated by pleasure in the toy material comes to look upon his parent as a desirable playmate and because of that begins to find satisfaction in communication that facilitates cooperative play

Demonstrations are provided from the lipreading cabinet and a sound filter system to show parents their children's need to combine listening and watching They are taught through personal experience that speech which they hear too imperfectly for it to be intelligible can be understood if they are given the opportunity to watch the face of the speaker while listening to him

Most parents seem to find it very helpful to hear recordings of filtered speech They are helped to understand what partial hearing is like, what their own children hear and what they miss To all, except perhaps a very few children, with the right kind of hearing aids, we can make at least some part of the sounds of daily life and of human voices and speech, audible On the other hand, it seems clear that there are no hearing impaired children whom we can enable to hear normally and completely even with the best available aids

(3) *Speech Training Aids* Experience of amplified sound is given to a child first of all by means of a speech training aid We loan many of these to parents Before doing so, we give the parents demonstrations and practice to help them to become skillful in using them effectively when on their own Guidance is needed for the parents about the length of time for which the aids should be used at one sitting Guidance in microphone techniques is essential, especially to help all the children who suffer from perceptive deafness to develop the voice to ear link

Apart from daily use of speech training aids, at all other times parents are guided to train their children to use wearable hearing aids continuously, from the time that they get up and put on their clothes in the morning until the time that they undress and go to bed

The vital importance of training children to accept and use spoken language as a matter of course in all the routine situations of daily life, is greatly stressed We ask parents to make sure that a little deaf child hears and lipreads "We're going out to the shops Let's put your coat on", as contrasted with mother just getting out the coat and putting it on her child

(4) *Wearable Aids* A child's first introduction to a wearable hearing aid should preferably be made at a guidance session in an audiology clinic. Parents need explanations of (i) care and maintenance of an aid (ii) methods of checking that it is working to capacity, (iii) the effect on speech transmission through the aid particularly in relation to the effect on sound of the distance of the microphone from the source and the influence of acoustic conditions and of background noise

(5) *Speech at the ear* The value particularly in certain circumstances (e g before issue of a wearable aid), of using speech and voice—singing talking—at the ear is explained and demonstrated

(6) *Parents voices and speech* Parents are helped to become critical of their own speech They need to speak with clear voices very distinctly and not too quickly

We find that it also helps parents if we make recordings of them and their children during a guidance session and then make a breakdown e g in terms of sentence structure and grammar They are helped by hearing the recordings replayed and the breakdowns discussed

(7) *Development of social habits in a child* Hearing impaired children need not be backward in such social habits as learning to dress or feed themselves Parents need to be aware of the standards of children who hear normally An outline of the stages reached by normal children at different ages is given to parents

CHAIRMAN Dr Lowell do you have comments?

LOWELL I would like to emphasize some of the points that I am sure Lady Ewing purposely did not include

## Group Meetings

I think that we must not overlook the importance of the psychological impact on the parents of discovering that their child is deaf For this reason the first meeting the first contact is a very poor time to try to get very much information across We ran an interesting follow up study in which we sent teachers into the homes of parents who had come to the clinic for an evaluation When the child was discovered to be deaf, the parents were given a great deal of what we thought was important and crucial information The follow up visit to the home showed the retention was about zero In fact I think that the first several visits are apt to be relatively inefficient for the transmission of information We find it is helpful however to have our class meetings with parents on a fairly frequent basis Even once a week is not as often as I would desire I think that the problem of parent education is one that has special difficulties when we approach it with the usual academic frame of mind We are faced with a group of adults and we tend to treat them like college students Unfortunately, that does not always work They come to us with different degrees of motivation and because their children are not all diagnosed at the same time they start

at different times during the year Therefore we must make our educational program a highly flexible one We must not be disappointed when we realize that our teaching efficiency is relatively low Parents who are not academically oriented may not like the classroom situation because of unpleasant memories of school Nevertheless we still find that it s helpful to put our meetings on almost a schoolroom basis with homework and with something in their hands to take home We find that if you put an observation sheet or a rating sheet or a note pad in their hands it helps them to get them back into the student role again

*Transfer to the Home Situation*

But the toughest job of all is to translate the school room work into the actual home situation We have now instituted a training program in a simulated home We have quite an unusual home It has three living rooms and two kitchens The teacher works with the parents in the simulated home situation while carrying out routine household activities The dishes for example get washed four or five times a day We also bring the other siblings the hearing siblings into the situation This creates havoc for the teacher at least but this after all is the situation that faces the mother We are finding that there is another level of training that we can offer parents namely informal language and the beginning of expressive language work before they are ready for the more formal instruction that they will do later

*Remote Families*

Our correspondence course is still of great value to parents living in isolated areas where they do not have direct personal contact with professional help A film training program a series of 19 motion pictures and recordings has proved a very valuable addition to the correspondence course A good picture *is* worth many thousand words and when parents can have something to read and work on in monthly installments and also a series of motion pictures that actually illustrates the material described in the correspondence course they have something of great value

We must always be aware of the great motivational value of having a group of people together who share the same common interest or common problem They themselves generate a great deal of enthusiasm and interest that helps our program enormously

Chairman Dr Griffiths would you like to add anything in the area of dealing with the parents?

*Care of Hearing Aids*

Griffith Yes Children are fitted with binaural hearing aids immediately as they start our program Therefore one of the first areas of education

of the parents is the care of those aids. We must teach the parents not only to put them on, but to check their performance before they put them on to know that they are in working order. We must teach the parents that a supply of hearing-aid cords and batteries must always be in the home, so that the child does not have to do without hearing at any time. *A mother must always see that she has the necessary equipment to make* those hearing aids functional.

We also instruct the parents that the child needs to wear his hearing aids all day long. When the parent notices that a child pays attention to sound she should direct the child's attention to the sound itself, as the child must learn to associate the sound with its source.

We ask the parents to talk a lot to the child and to play with, talk, and sing to the infants.

We have parents' meetings which provide group therapy. Fortunately, the children come regularly once or twice a week for lessons which provides the opportunity to answer questions of parents during these sessions.

## *Discussion*

SILVERMAN: We must not underestimate the impact of one parent on another in these group situations. In order to study this we had a person trained in this sort of thing attend all of our parents' meetings and take transcripts. We excluded from the meeting the audiologists and "parent-trainers". There was quite a ventilation of attitudes such as frequently doesn't come out in the interaction between the professional person and the parent. The parent's attitude toward the child's deafness very frequently is revealed in the parent-to-parent context but it may not get revealed in the teacher-student situation. We have a very interesting transcript of ten such meetings and have just been having some independent person analyze it.

For the past six years we have had an active program for pre-nursery *hearing impaired children and their parents under the direction of* Dr Audrey Simmons. Our experience suggests that fundamental for the help to the child by the parent is the parent's understanding of the problem and process of language acquisition by the child. The main point in convincing parents of the importance of their obligations and opportunities is to emphasize that they are the major, if not the only, source of language input in the early part of a child's life. It is not only the child's capacity for hearing that determines his acquisition of language, but also how much language he has an opportunity to hear. The critical factors are hearing *and* talking. We have found that the display of steps in language development shown in Figure 7 is helpful in getting across this idea. Of course, our presentation accommodates to the educational background and attitudes of the parents. For example, we may take the parents through the steps by beginning with items of language that are unfamiliar to *them*

SOME STEPS OF LANGUAGE DEVELOPMENT

SKILLS

MATURE LANGUAGE

CONNECTED LANGUAGE

LARGER EXPRESSIVE UNITS

LIMITED EXPRESSIVE LANGUAGE

IMITATIONS

FREE COMPREHENSION
CONCEPTS
CONNECTED LANGUAGE

SITUATIONAL COMPREHENSION
CONCRETE ITEMS
VISIBLE ACTIONS

AWARENESS
CONCEPTS
VOCABULARY

EXPOSURE

Fig V 7

Good suggestions, for obvious reasons, would be words like 'decibel', "sensory neural' and "binaural"

We then show how the steps apply to babies Right at birth and perhaps *in utero,* as we have heard at this conference, the child is 'exposed' to talk Nurses and parents are the first teachers' of language Following this 'awareness" is helped along by labeling objects, by talk activities and key words take on meaning by repetition Gradually, experience in situations moves the development a step further Mommy has on her coat and hat, and she tells Johnny to get his and they ll go out The situation calls for performance, and we begin to achieve 'situational comprehension" After this, he may get the coat before mother has made any effort to get hers (free comprehension) This leads to spoken 'imitations' like 'mother-coat" In a normal hearing baby, this may occur at about 18 months Here we stress that the child *has had to do a good deal of listening* to get to this stage It is these early stages of development that we discuss in great detail with parents Of course, the later stages that mark logical objectives are cited, but their elaboration is left for subsequent periods in our parent program

We are now initiating investigations to study the effectiveness of our procedures on language performance We believe that a satisfactory description of language is essential for an improved understanding of how language is learned and, consequently, how it should be "taught" Investigators and teachers have sought diligently for descriptions and measures beyond those yielded by standard tests They have not been satisfied by vague and frequently misleading assertions about deaf children being 'two to five years retarded' or about their having typical "deaf language' They have used

such measures as sentence length and complexity, frequency of occurrence of certain parts of speech and word order, extent of vocabulary, type-token ratios (the ratio of different words to total number of words used in a written or spoken selection), subordination, and abstractions They have used the methods of structural linguistics to analyze the functional and lexical features of the spoken and written language of deaf children.

We are recording the spoken language of our young children and trying out some schemes for quantitative description Of course, this is in addition to the usual narrative and performance on certain conventional tests

BARR: The prerequisites for guidance of the parents of deaf and hard of hearing children are intensive instruction and training combined with a thorough medical, audiological and psychological investigation. Extremely few of the deaf or severely impaired children are "pure" cases of deafness Many of them have complicated handicaps such as defects of other senses or behavior problems The medical-social investigation shows that the economic and psychic stress on the family is too great to create the natural atmosphere that a deaf child needs so badly Everything that can be done to improve this situation should be done in addition to all the other measures The chief aim should not only be a child that speaks as much as possible, but primarily a happy child And the parents should feel the satisfaction of having been able to contribute to this result

When the child is subjected to its first examination at Karolinska Hospital (Stockholm), the parents immediately receive concentrated instruction about everything regarding the care and education of deaf and hard of hearing children At first the talks are of a general nature and refer to matters that also apply to children with normal hearing When the examination of the child has reached the stage of a well-established diagnosis, the talks are more and more conducted so as to make the parents accept the fact that their child is deaf or hard of hearing The parents must learn the basic principles of the training of senses, hearing and lip reading. After this theoretical preparation the parents visit the kindergarten for hard of hearing children They watch the practical work and are advised about the purchase of suitable teaching material

For children in Stockholm and suburbs, home training is organized so that teachers from our institution regularly call on them once a week or once a month as may be needed The parents meet every month in large or small groups and discuss their problems with one another and with experts

The contact with parents who live far away has been kept up by letters telephone calls, and regular visits to the institution Some time after the first investigation the parents are given the opportunity to participate in a one week course Sometimes these parents, who often live far away from large communities, meet others with the same problems for the first time

Their own children are not with them during this week and other deaf children are used for demonstrations

Nowadays most of the children are examined at such an early stage they are much too young for attending a kindergarten But even if the child is old enough to attend a pre school, the parents have to try to take care of the child themselves for some time after the first investigation Immediate schooling has been recommended in only a very few cases where the condition of the home was such that no help could be expected from the parents

Experience has shown that parents whose child has been placed in a school immediately after they have received careful instructions are apt to turn over the responsibility for the education and training of the child entirely to the school Parents who have worked with the child themselves for one or several years, however, know much more about all its problem and take an active part even after the child has started school

All this is paid by the county or the state From the first of July this year parents in Sweden receive 2500 kr (\$500) allowance every year if they have a child with a severe handicap, as for instance deafness

WEDENBERG I would like to go a little deeper into the instruction of the parents in hearing training Our parents are instructed in the physical structure of speech sounds, on the formants, on the frequency ranges in which they lie, on their intensity in speech *ad concham* and in speech at other distances They are given a formant table consisting of an audio gram sheet on which the Swedish speech sounds are entered with their intensity in speech *ad concham* On this sheet they draw their child s audiogram and they can then form a picture of the sounds that they should train, that is, the sounds that the child hears and also those that the child does not hear and that they should not train for the time being The parents are also given a list of words that are suitable for practising

A question that should be discussed in connection with the selection of words concerns the first names of the pupils Many of them with grave auditory impairment in the "high frequency range" had first names they could scarcely hear, even in speech *ad concham* Among these are the names Chris, Christer, Stig Kerstin, and Staffan A child who does not hear his own name, either when others say it or when he attempts to say it himself, has a rootless existence Therefore it was necessary to exchange these "high frequency" names for other "low frequency" names that the children could perceive If a child had several Christian names, the one with the "lowest frequency" was selected, e g Anna, Valter, or Douglas Otherwise, the child was "rechristened" As an illustration of this problem, one boy whose Christian names were Christer Valter, and who was called Christer by his parents, decided without outside suggestion to call himself Valter, despite his parents' use of the other name

HUIZING In 1961, at the second International Course in Paedo-Audiology held at Groningen University, I had the opportunity to report on the case of a deaf child, which was given home-training from a very early age He suffered from meningitis at the age of three months Deafness was suspected at about six months Daily use of a wearable hearing aid was prescribed when he was sixteen months old His mother was given audiological instructions

This child responded very favorably to the home instruction At the age of eight and a half he has now an active vocabulary of about 1500 words In school he is doing very well His hearing function is characterized by total deafness in one ear and 80 to 100 dB loss (ISO) in the other ear, the upper frequency limit being 2200 cycles per sec

Even more interesting is the case of a young boy whose deafness was caused by maternal rubella The handicap was suspected for the first time at the age of four months He didn't react to singing as his one-year-older brother at that age His mother got the impression he was unmusical, he didn't try to find the source of all kinds of stereotyped sounds Being looked at in the cradle often made him start Auditory contact was only possible for his resourceful parents by means of a loud voice through an improvised conic cardboard hearing-tube A wearable hearing-aid was prescribed, the parents were instructed about its use with the aid of literature, etc At the first tests the child wasn't reluctant, application of the hearing-aid in its concha went without difficulties Switching on the apparatus at low volume setting didn't bring about notable response, but at high amplification there were unmistakable reactions up to 1200 c/s or even higher up in the pitch range For that reason we advised the parents (to whom we are indebted for their minute reporting) the daily use of an apparatus on both left and right ears at the same time, at a high volume setting

The first day at home no positive reactions could be observed, the second day he showed feelings of discomfort The third and the next days *clearly* proved sounds perception by way of his mimics

*At the eleventh day he was startled by sound and his mother awoke him* by clapping her hands Depicting the situation would be as follows the child has fallen asleep, still wearing his hearing aid When, at a half *metre* s distance from the microphone, his mother claps her hands, he awakes with a start This game lends itself for repetition Without a hearing aid it wouldn't have had any result whatsoever

From the first day of "hearing" his parents used to attract the boy's attention by whistling (father) and mother clapping her hands

On the 17th day he appeared to be conditioned, that means whistling will make him look at his father, clapping at his mother We are *thankful* to the parents for their accurate observation and data

Very soon the hearing-aid proves to have become something belonging to him the receiver in his concha isn't a strange contraption any longer but a corporally integrated part It belongs to him inseparably

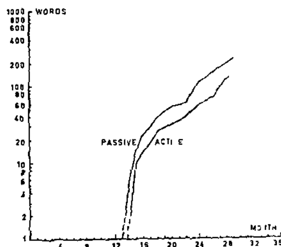



Fig \ 8 Growth of vocabulary (active and passive)

In the fourth week of hearing it becomes apparent that the child tries to find the source of sounds, in the fifth week it obviously makes him glad that the apparatus is switched on

From that time a vocal play is developed with which, by means of the microphone, he addresses himself, a kind of "a ' babbling This vocalization is instrumental to his becoming aware of his personality The hearing remnants are developing into an auto excitative and controlling function

Summarizing, it may be said with certainty after three months that with his hearing aid the child adequately reacts to sounds such as whistling, clapping hands, and humming, that he tries to find the source of sound, and that he looks at his parents when spoken to So at the age of nine months he has acquired facial attentiveness, has learned to listen, and an auditorily controlled voice is being developed

After having learned to listen, learning to understand must commence, which means learning to discriminate speech sounds in order to be able to generate language

Continually recognizing familiar sounds leads to an increased feeling of safety and encourages the child to conquer the space around him He starts crawling and walking and at the age of one, he more or less keeps his balance When about twelve months old he adequately reacts to the conception meaning of the word "no' At the age of fifteen months he listens to his name and when somebody calls come here', he leaves his game and does so He has now arrived at the point where he begins to call objects by their names, and this is systematically promoted as much as possible

Meanwhile towards the end of the second year, the parents have been advised to enlarge the auditory training apparatus with a battery-operated hi fi table amplifier It is regularly used The child wears the double

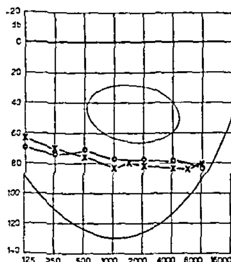



Fig. V: 9. Threshold audiogram of a case of very early treatment (cf. Fig. V. 8) by home-training from six months on (ISO scale)

headset, both at play and at dinner. Systematic training with this apparatus takes place; rhythmically constructed sentences are much used. Active speech has commenced at about the age of one; now, when two years old, he pronounces about 50 words. His speech typically consists of vowels. His voice sounds quite right; it is not monotonous. He has used two-word sentences from the age of two years and two months; three-word sentences when two years and four months old.

The initial phase of the child's linguistic development is shown in Fig. 8, which shows the gradual growth of his word vocabulary. Fig. 9 represents his threshold audiograms, covering the whole pitch range, so that this case may be considered as a favorable one from the viewpoint of residual hearing.

From the above mentioned practice the conclusion may be drawn that in certain cases of severe hearing impairment, prevention of deaf-mutism through prompt acoupedic treatment is attainable under certain conditions. These conditions are:

1. Early detection of the handicap;
2. Adequate auditory approach through home-training should be initiated as early as possible, preferably before the first birthday,
3. Emphasis should be laid on the importance of a favorable linguistic milieu and on quiet surroundings;
4. Audiological instructions of the parents tuned to the particular case of each child,
5. A special pre school, corresponding to the audiological needs of each child, at the age of three or four.

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CHAIRMAN This is a very interesting and informative case Would you tell us again at what age formal auditory training and systematic drill was begun?

HUIZING It began at about age two

CHAIRMAN How many hours a day, more or less was devoted to this? How much can a youngster of this age tolerate?

HUIZING The drill was done in connection with meals perhaps three or four times a day We just gave the parents instructions as to what type of linguistic material would be used

CHAIRMAN Do you think the result would have been any better if you had started at three months instead of at six months?

HUIZING Well, I don't know

### *Home Visits*

RØJSKJAER In my small country we also have the parents visit the clinic very regularly We know that it is very important for them to meet one another to talk together about their problems, but the major work in Denmark is done by regular visits to the children's homes by trained consultants or teachers throughout the year They visit the children and their parents every month or two all through the critical years Perhaps that's not possible in every country

A EWING We have a number of parents who have been found by trained psychiatric workers to have some problems of their own, often emotional, sometimes relating to their early history, a deprived childhood, and so on The information that the trained and experienced psychiatric worker can bring back is extremely helpful to the "guiders", as we now call them We would like to get far more of the guidance to take place in the home, but it is a matter of arithmetic the case-load in relation to staff that can be financed and made available

BARNET. An accumulation of evidence from several fields gives support to the concept that sensory deprivation early in life causes impairment in the organism's later functioning which may be permanent. Much of the work has been done in the conceptual framework supplied by Hebb (1949), who stressed that early learning which takes place through the senses provides the necessary basis for thought processes which later develop. The difficulties of patients after the removal of congenital cataracts in learning to see were described by Von Senden (1932). Their progress was often discouragingly slow and even after many years they might make mistakes, as in the task of rapid identification of a series of objects presented very briefly.

This paper will briefly review several kinds of work: behavioral, physiological, anatomical, biochemical, on the effects of sensory deprivation.

Most has been done with the visual system for several reasons, among which is relative ease of providing a situation of temporary deprivation. In the typical experimental setup, the animal is either raised in a dark room, or his lids are sutured, or plastic occluders are used to diminish light and or pattern stimulation. The fact that normal visual anatomy and physiology are relatively well understood also explains the greater work with the visual system.

In 1937 Hebb reported (1937 *a*, 1937 *b*) an experiment in which 18 rats were reared in darkness until they were 60 days old. They were then subjected to a situation where discrimination of horizontal from vertical lines was necessary. Normally reared rats took about 20 trials to learn this discrimination, but the dark reared rats averaged 129 trials. After a time, however, the dark reared rats appeared to act like normal animals; thus the effect of 60 days of visual deprivation was apparently temporary.

Riesen and his associates have performed a series of studies (see Riesen, 1955, 1961, also Hunt, 1961) on primates who were reared under conditions of light or pattern deprivation of varying duration. In one experiment, three chimpanzees were placed from birth in a dark room for seven months. One remained in darkness for the duration of the experiment. The second was permitted one and one-half hours a day of light diffused through a plexiglas dome attached to his head. A light was turned on over the crib of the third chimpanzee for one-and-one-half hours a day. He thus was able to see the form and movement of his own body, and the shape of the feeding bottle and his crib.

At seven months the three animals were tested in the light. The dark and diffused light reared subjects both showed severe disturbance of visual behavior although pupillary light reflexes and head turning towards bright [illegible]ht were present from the beginning. A normally reared seven-month-old [illegible]e can be expected to show smooth visual fixation, to open his [illegible] his feeding bottle as he sees it approach, and to fear strange objects or persons. These reactions were initially absent in the dark and

diffused light reared animals and appeared only after six to thirty days They appeared less rapidly in the dark than in the diffused light reared animal. When subjected to various visual training procedures, these chimpanzees required very many more trials than normal animals In contrast to the first two chimps, the third reared with one and one-half hours of patterned light daily, was almost indistinguishable from the normal animal in his visual reactions and his reaction to visual training procedures

Some maturation must have occurred in the absence of visual stimulation because the dark reared animals took only from six to thirty days to acquire visual reactions which a normally reared chimpanzee does not acquire until about two months of age

Experiments using *monocular* deprivation have confirmed the above results The animal is trained to a visual task using the eye which had received one-and-one-half hours a day of normal sensory input Ordinarily a discrimination learned with one eye is readily transferred when the other eye is used If this eye, however, had received from birth only diffuse light stimulation, the animal could not perform successfully using that eye except after many trials The magnitude and permanence of functional effects after deprivation appear to be related to factors including the age when the animal is deprived, the extent and duration of deprivation, and perhaps also to the relative presence or absence of stimulation through other modalities (see below, p 213) It seems clear that deprivation early in life causes more deficit than the same length of deprivation in the older animal In addition, the type of deprivation, whether of light and pattern or pattern alone, appears to have differential effects

Some of the observed behavioral effects such as failures in learning or fear of objects and persons, which have been ascribed to the lack of a specific sensory input, may be influenced also by much more complex factors leading to disturbances in affect or level of responsiveness For example, dogs which have been isolated early in life in a dimly lit but not patternless environment do not learn simple visual discriminations as well as normally reared dogs, although they have not been completely deprived of pattern vision (Melzack, 1962) Their reactions to painful stimuli are abnormal, for example, they do not yelp or move away when touched with a burning match or jabbed with a needle They also show violent emotional excitement in unfamiliar surroundings

Physiologic correlates of visual deprivation have been explored in studies by Hubel and Wiesel (1963 *a*, 1963 *b*) which have given convincing evidence of specific impairment in the visual system Kittens were deprived at the time of normal eye opening by suturing the lid of one eye or by covering it with a translucent occluder The occluder let most of the light through but deprived the animal of patterned light, lid suture, however, decreased the amount of light by about 4 log units After two to three months of deprivation recordings were made of the activity of single neurons in the visual cortex in response to patterned light The experimental procedure,

briefly described, is as follows Microelectrodes were introduced through the skull over the striate cortex of the anesthetized cat who was positioned facing a screen on which different patterns could be projected One eye was covered The orientation of the pattern in space and the part of the retina on which it was projected were slowly changed until the cell with which the electrode was in contact began to fire After the characteristics of the cell with respect to the retinal field it was influenced by, and the orientation of pattern it preferred were noted, the first eye was covered and the second eye uncovered In the normal animal when the corresponding retinal field of the other eye is stimulated the cell will respond This is true also for the normal kitten at and before the time of eye opening (Hubel and Wiesel, 1963 *b*) About 85% of visual cortical cells are thus binocularly influenced although a stimulus on the side contra-lateral to the cell being recorded from, drives the cell most readily After the characteristics of the cell in response to stimuli from each eye were noted, the electrode was advanced slightly and the above procedure repeated In a single penetration which took many hours, 20 or 25 cells could be mapped The track of the electrode was marked with electrolytic lesions and later histologically verified

As stated above, in the normal cat at all ages cortical cells could be driven by stimuli presented to either eye In the monocularly deprived animal, however, they were driven only from the eye which had received normal stimulation For example, only one out of the 84 cells, on which records were made, was influenced by the deprived eye A few cells could not be driven from either eye and were identified only by their spontaneous activity The effects of monocular pattern deprivation without light deprivation on cortical physiology seemed to be as severe as those seen with both light and pattern deprivation

Even with the severe disruption of cortical function, cortical morphology studied using H & E and cresyl violet sections showed no definite abnormality

In the lateral geniculate body, however, which is the last way-station for visual impulses before the cortex, electrical recording from cells receiving input from the deprived eye showed relatively little disturbance This indicates that the inability to drive the cortical cells from the deprived eye was most probably due to a defect in the cortex itself A surprising observation in view of the relative normality of lateral geniculate physiology, was the marked histologic abnormality of that structure The layers receiving projections from the closed eye were thinner, because of a loss of intercellular substance and because the cells themselves were shrunken The anatomy of the LGB was much more severely affected in the kittens who had received light as well as form deprivation, which is in accord with [illegible] fact that geniculate cells normally respond most actively to change [illegible] light illumination

It is noteworthy that the effects of *monocular* deprivation appear to be

relatively specific to that situation When physiologic study of visual responses was made on cats raised from birth with *both eyes covered* (*Hubel* and Wiesel, 1964) it was found that while two-thirds of the cells either did not respond at all to stimulation from either eye or were abnormal in response, one-third of the cells were entirely normal Thus disturbance of *neural connections* with monocular deprivation are apparently more profound than those seen with bilateral sensory deprivation In both preparations, however, functional damage was severe The animals appeared to be blind in the deprived eye or eyes and there seemed to be little recovery even after months in the light

Evidence for structural damage at a level below the LGB is given by Brattgård (1952). In rabbits kept in darkness for ten weeks, the retinal ganglion was found to have reduced cell density and a great reduction of the protein content of the cells, notably the pentose-nucleo-proteins Similar effects have been found in chimpanzees (Chow, Riesen and Newell, 1957) The electroretinogram of deprived animals appears to be relatively normal at least after several *hours in the light* (*Zetterstrom*, *1955*, *Baxter and Riesen*, 1961; Hubel and Wiesel, 1963 *a*), indicating probably that the receptors themselves are relatively undamaged by deprivation

Effects of visual deprivation on brain acetylcholinesterase activity in rats have been described by Krech (1964) who compared cholinesterase activity of the visual and somesthetic cortex and subcortex in litter mate rats who were either dark reared for 100 days, normally reared, or blinded by enucleation At 100 days the animals were sacrificed and the brains studied (for a recent review of this work see Bennett, Desmond, Krech and Rosenzweig, 1964) The results in blinded and dark reared animals were similar the weight of the visual cortex was less by about 4% than that of the normally reared animals and the cholinesterase activity was significantly greater per unit weight, also by about 4% The neurochemical change was therefore felt to be the effect of stimulus deprivation rather than anatomical changes resulting from enucleation Dark reared rats who were kept in a large cage with other rats and who were handled frequently were found to have a larger somesthetic cortex than the dark reared animals who were kept isolated in small cages Differential effects in cholinesterase activity were seen in rats raised in varying degrees of social isolation and enrichment The investigators concluded that complexity of environment is an important determiner of brain cholinesterase activity The differences seen are small and may be within the error of sampling and analysis (Tower 1958) The finding of a neurochemical substance known to be important to CNS function whose concentration is sensitive to environmental factors is however, of great significance

In one of the few experiments concerning deprivation of auditory stimulation which has been done, behavioral effects which are analogous to those seen with visual deprivation were found In 1943 Wolf deprived one group of rats of hearing and another group of vision for a ten-to fifteen day

period in infancy After the rats had reached maturity, they were compared in performance tests where response to a buzzer or a light was the signal The rats which had been visually deprived in infancy did less well in tests requiring response to a light than a buzzer Conversely, rats deprived of hearing performed less well in the test with a buzzer as signal This experiment was replicated and the results confirmed by Gauron and Becker in 1959 who interpreted their findings to mean that a specific deprivation caused a limitation of sensory ability in that modality It was found however, with repeated testing that the performance of the two groups of animals gradually ceased to differ significantly

*Summary*

A picture of the importance of sensory stimulation for brain function emerges from the work of investigators of varied disciplines In order for the perceptual requirements of the organism to be fulfilled sense data must be available in the same way as essential dietary components for physical nourishment There are perception deficiency states which come about through mechanisms analogous to those seen in vitamin deficiency diseases The relatively greater effect of early vs later deprivation is noteworthy, *but the question of the permanence and functional importance of* defects is still unclear The finding from physiologic recording that responses from the newborn animal are similar to those of the adult suggests that the neural connections do not require stimulation for development but are innately determined Absence of sensory stimulation then, would cause a deterioration through disuse Although some of the newer work is exploratory in nature and needs to be confirmed and extended, the findings have important clinical implications in the management of the young child with sensory handicaps

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DAVIS Dr Barnet as you point out the work is still quite recent but the Hubel and Wiesel experiments have at least gone far enough to point to clear anatomical and also physiological changes and to indicate that these are long lasting if the deprivation begins very early

WALTER I think there is no doubt that for deprivation of visible light to cause a lasting effect the deprivation must be both early and complete This is certainly true for pattern vision Apparently the sensory system continues to function more or less normally if it is activated by any means Stimulation of a few sensory fibers would be enough Of course there are no studies of this kind in human deaf children but we have extensive ex perience with human blind children who suffered from retrolental fibroplasia These children are now at least 12 years old (I don t think we poison premature babies any more but this turned out to be a very good control experiment ) These children were totally blind from birth and there is no doubt that there is gross and widespread abnormality of the occipital cortex both in the primary and in the association areas Other interesting features are the lack of EEG rhythms and the appearance of

EEG abnormalities. All this is certainly good evidence that total congenital deprivation does produce effects in man.

Now whether auditory deprivation could produce a long term effect is a matter of conjecture. It seems to me rather doubtful that this would be a very important factor in regard to the management and assistance of deaf children, certainly unless the loss was so great as to cause almost total deafness. By this I mean complete failure of the brain to be aroused by any unamplified sound at all. In such extreme cases I would say that the assistance provided by even the simplest hearing aid used in the earliest days, would be invaluable, but I wonder whether one shouldn't be a little cautious about simply raising the noise level. I think it would be rather clever if we could arrange the hearing aid to emphasize intricate sounds such as speech *rather than all sounds. A voice-operated—i.e., voice-activated*—device might be used at first. Later in life a manually operated instrument would allow the child to learn to turn the intensity up in response to significant signals such as seeing a mouth moving or a face with an expression on it. This selective sensitivity might be quite important, giving emphasis to a patterned sound rather than to noise. You will remember in the Hubel and Wiesel experiment they found degeneration with eyes that were allowed to see light but *no patterns. The inference* is that we might not help much by simply raising the total level of stimulation by random *noise*.

WEDENBERG: Through the investigations by Brattgård (1952) on retinal ganglion cells of rabbits born in darkness and living the first ten weeks in darkness we know that the nucleo-protein fraction in these cells was absent. The control animals living in diffuse daylight had a relatively high nucleoprotein fraction. The experimental animals had perception of light and some *degree of discriminating vision. Another of his animal groups* also born in darkness and living the first ten weeks in darkness was exposed to daylight for three weeks. The nucleoprotein fraction *then rose*. This shows that in connection with the onset of adequate postnatal stimulation, the nerve cell enters a period of chemical growth. The lack of adequate stimulation during the early postnatal period results in incomplete development of the nerve cells and to disturbances in their metabolism.

These investigations explain clinical observations on children. If one eye is excluded from adequate stimulation by light, for example, through *cataract or a bandage*, the *visual acuity decreases and amblyopia* develops. This is not the case when a cataract develops in an adult, for when the cataract is removed, the eye will function adequately.

In the auditory system Hamberger and Hydén (1945) have investigated ... cytochemical changes in the cochlear ganglion caused by acoustic ... sound trauma. They have found that moderate stimulation leads ... the nucleoprotein fraction of the cell. There is simultaneous production and consumption of nucleoprotein in the cell. There is, how-

*Acta oto-laryng. 206*

ever a great difference in early stimulation and development between the normal vision and the auditory system The retinal ganglion cells are the only nerve cells that can be entirely excluded from stimulation experimentally In normal development the auditory ganglion cells are never deprived of stimulation either before or after birth During the 14 or 15 last weeks of pregnancy the foetus has an auditory stimulation from the mother s heartbeats and intestinal sounds it is therefore probable that nucleoprotein is produced in the newborn owing to the foregoing stimulation If a hard of hearing child grows up without auditory training or becomes too dependent on speechreading it never learns to listen Hence if there is a lack of stimulation during the early postnatal period the development and chemical composition of the nerve cells can be retarded I therefore begin the auditory training as soon as I have made a diagnosis of hard of hearing —even in the first month of life A child is never too young to listen

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Davis The human central nervous system is not fully developed at the time of birth It is much more nearly mature at two years but even here there is no sharp dividing line However we know that at two years of age some of the tracts of the central nervous system are not yet completely myelinated and for some other parts of the nervous system we simply have no idea when this myelinization may be complete and the structures ready for normal function

There seems to be some pretty clear clinical evidence that the central nervous system is more plastic in the first two years of life than it is later By plastic I mean that following injury there is better compensation by some kind of reorganization or by a different pattern of organization The clinical evidence is that equivalent injuries are better compensated for if they occur early An example is the determination of which will be the dominant cortex whether for handedness or for speech But in either case it seems as though the two cortical hemispheres are originally equipotential and then one side usually the left is picked out as it were and manual skills and the understanding and the formulation of speech are organized on that side If there is early injury to the left hemisphere then the right hemisphere takes on this function but the transfer is not made in later years This I mention as evidence for a greater plasticity of the nervous system in the first two years

Whetnall There is evidence that dendrites may continue to proliferate in the human cortex into the late teens If this is so we may hope that the

nervous system remains highly plastic until the late school and even the early college years I mean plastic in a physical sense, quite apart from more subtle functional variations Of course this is not a direct experience of my own but it is the point of view of some histopathologists, such as Paul Glees in Germany.

DAVIS Would you care to associate this continued growth of dendrites with ease of learning?

WALTER Yes, I would be tempted to do so, and also to associate it with the contingent negative variation (CNV) which I described yesterday This electrical response tends to grow bigger and neater up to the age of 20

In regard to the lateralization and location of hearing and speech centers, Penfield and Perot (1963), in their fascinating monograph, plot all of the cases in which electrical stimulation of the cortex produced auditory sensations or modification of speech. The distribution is extraordinarily widespread as compared with the classical maps in textbooks which may identify only Broca's area

In the cases of temporal lobe disorders we encounter a great diversity of interference with function I think these disorders must contribute to the difficulties of those people who find it hard to associate meaning with sounds or to learn to formulate speech These cases of early disorders of the temporal lobe are subtle and difficult and I believe that they are one of the exceptions to your rule, and that is why I brought it up The young nervous system is plastic, but nevertheless early injury to the temporal lobe produces long-lasting disturbances which are very hard to rectify Perhaps we should not be too optimistic about them The temporal lobe presents a special case with its very complex functions and its marginal blood supply

DAVIS And possibly its very close association with the hippocampus also That is a rather important structure just beneath and medial to the temporal lobe

RAPIN In terms of lateralization of speech there is some plasticity in early childhood I would like to report the case of a five-year-old child, in whom it was thought, on the basis of amytal tests, one hemisphere was dominant Because of major epilepsy, it was decided nevertheless to perform a hemispherectomy on that side To everyone's great surprise, the child was not even transiently aphasic after the operation, indicating that although the amytal test is certainly quite useful in adults for identification of the hemisphere which is dominant for language, it should be interpreted with caution in children as young as five years

DAVIS Perhaps the lateralization in this child was not yet solidly established one way or the other

HAWKE The most interesting point is that the plasticity diminishes as the child grows older. I think that shifts of the language center are very rare beyond five or six years of age Another interesting point is that the newborn animal is primarily a 'brain-stem individual" I think that the reason for the importance and permanence of early temporal lobe damage is that it represents a different functional level At some early stage of our development we are functioning primarily at the hippocampal level Remember that when we talk about plasticity no one quite knows whether what happens may be that our primitive centers remain and continue to funcfion In other words, if our cerebral hemispheres do not develop properly, the functions may remain in the primitive hippocampus Following failure of cerebral areas the function does not necessarily, as some people think, shift to the intact cerebral hemisphere I find very interesting the idea that the degree of plasticity varies with the area, in respect to both its microscopic structure and its functional organization

LOWELL (afterthought) Dr Grey Walter suggested to me that it was remarkable that we have not mentioned the Russian literature on the development of the "second signal system" I think we should have on record at least a reference to Luria's work This system has been extensively studied by the Russians and their studies may well contribute to this problem of the critical period in the development of sensory systems

WALTER In Moscow there are quite a few scientists including Luria and his colleagues working on defects of all types They have an extremely useful mass of data that we Westerners have not tapped at all It has been published and we should read it Some of it is of the highest quality

DAVIS I think that out of all this comes the suggestion that until a certain degree of maturation has occurred in the cortex it will probably not make much difference whether there is sensory deprivation or sensory enrichment What we need to know is exactly when it is necessary to move in and provide sensory input in other words, to know when the developing brain is ready to take advantage of the input At present we rely entirely on intuition, and for the present perhaps that is the best we can do Perhaps clinical evidence will give us some additional insight

## F. BENEFITS FROM AMPLIFIED SOUND

### *Clinical Evidence of Benefits from Amplified Sound*

CHAIRMAN Dr Whetnall, you employ amplified sound with hard-of-hearing children from an early age Will you open the discussion of its benefits?

WHETNALL: I have selected three children, for you to see their audiograms and hear their speech I saw each of them some years ago and therefore by now their speech is well established Our decision to train babies and our methods of teaching them follow the lines just described by Professor Huizing The whole method is so simple that there is no question but that this is the right way to do it

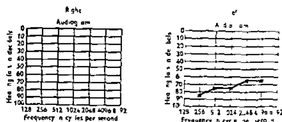
The first figure is the audiogram of a boy whom I saw in 1948 when he was six years old He had meningitis at the age of one, and at the age of two he was given a hearing aid *The point I emphasize is that he had* good speech when I saw him and also excellent comprehension for his age (Dr Whetnall illustrated his speech with a tape recording) I continued to see other children in that year who had spontaneous speech in spite of very severe hearing losses, but in every case in this group of children with good speech the mother had suspected the deafness early By early I mean in most cases before 18 months and sometimes during the first year, and she had talked close to the child's ear. The type of loss in each of these cases was variable, high-tone loss or a flat audiogram or an audiogram rising toward the high frequencies The severity of hearing loss was such that if they had been seen before their hearing had been trained, they would undoubtedly have been considered too deaf to be trained this way The idea was ingrained at that time that the hearing of the deaf child was useless We concluded, however, that it was neither the type nor the degree of deafness that prevented the deaf child from learning to talk but some faulty idea that people had about the use of hearing

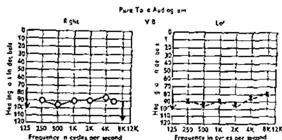
Children were next selected for auditory training whose loss of hearing was comparable to those with spontaneous speech Their hearing was assessed clinically as some of them are too young to do a pure tone audiogram and it was already my opinion that one could not predict or assess the child's future ability to use his hearing from a pure-tone audiogram I selected them by their ability to respond to my voice at one foot and sometimes directly at the meatus

Two effects of extreme importance were established in this next group First, the child originally could hear but could not understand, but after a period of training, with continual practice which might last up to a year or 18 months, the child started to understand words and sentences and at about the same time or a little later would start to use words This change had of course, already occurred in children with spontaneous speech and it indicated that learning was taking place

The second important point was the age of the child The younger the child the easier the progress The young child likes his aid, he likes sound and will not in any case be without his aid

It seemed reasonable that the young deaf child should be allowed to *hear at the age at which a normal hearing child learns* and quite unnatural that he should not be given this opportunity The onus of proof is on those who wish to delay learning in the deaf child until a later date



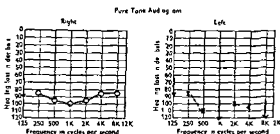




Fig V 10 Deaf from meningitis at age of one year 1st case on tape (Br )
Fig V 11 Deaf from maternal beri beri 2nd case on tape (Br )
Fig V 12 Deaf from meningitis at age of 15 months 3rd case on tape (Br )

The third figure is the audiogram of a boy who had meningitis at 15 months before he had developed speech He was immediately taught to listen and given auditory training continually by his mother

A common language must be present if communication is to take place There is a vast store of linguistic knowledge on which speech depends Every language is a system of units of varying sizes Linguistic knowledge consists of knowing what these units are, how they function and how they combine Knowledge of the way in which they combine is stored in the memory of every speaker and every listener, not only the way in which they combine but also the statistical probabilities of their combinations These facts about a language are stored in the brain where we pay little or no attention to them and are usually quite unaware that we possess this knowledge The most important asset for speech recognition is knowing what to expect We do not have to hear everything If we hear half we fill in the rest by guesswork and never make a mistake

Speech may be understood in the face of all kinds of interference Fry states that all of the more recent discoveries of the nature of speech reception suggest that there is no reason why the deaf child should not succeed

in learning to speak. It is not true that speech cannot be developed unless the child has hearing for such and such a frequency band, nor is there a particular indispensable version of the sounds of the English language without which the production and reception of English speech is impossible. The truth is quite the opposite; we know that the perceptions of the deaf child are different from those of the hearing child and that the perceptions used in speech are all learned. The normal listener has a redundancy of cues available to him. There are many cues that can be used, and the deaf child will learn to use all of those that lie within his range of hearing and if necessary will develop his own cues to replace those that are missing. We hear with our brain, and it is the extraordinary capacity of the brain to organize information which enables a deaf child to learn and to use the cues which the brain receives. The deaf child will do this only if he is given the same opportunity as the hearing child, the same encouragement, and the same facilities for learning. The sounds must be heard loud enough, preferably with two aids as children have two ears. Often enough, during *the first years of life, the child must be given individual attention by* some one. It is usually the mother who does this work, and without it the child will not succeed.

CHAIRMAN. Thank you, Dr Whetnall. May I ask you to recapitulate the changes that you observed in the audiograms of these children? Did their peripheral auditory sensitivity change?

WHETNALL. There were three different children. One of them was two-and-one-half years old before I saw her, and there was virtually no subsequent change in this audiogram. Another child was four years old when the audiogram was made. He is now eight-and-one-half years old. His hearing has varied a little as a result of a conductive deafness produced by infection of the middle ear, but the audiogram that I showed represents about his usual level. The first audiogram to be taken of him showed a 100 decibel loss.

DAVIS. 100 decibels? Is that American Standard or British Standard?

WHETNALL. It is British Standard.

DAVIS. Nevertheless I was surprised at the flatness of the audiograms. We usually see more of a remnant of "hearing" down in the lower frequencies.

GLORIG. Dr Whetnall, have you had similar experiences with children whose hearing spectrum terminates at 1000 c/s?

WHETNALL Yes There were two in this first group They had spontaneous speech but it was partly defective I have subsequently seen children with high tone losses sent to us because someone had done an audiogram at school but nobody up to that point had suspected that the child was deaf— not even the parents in one case

GLORIG You are talking now about 80 and 90 decibels at 500 and 1000 cps and no hearing beyond this?

WHETNALL Yes Two weeks ago I saw a child aged four years whose deafness had been detected by a speech therapist six months previously This child s speech was obviously a little defective and that is why she had gone to a speech therapist Her hearing loss ranged from 60 dB (British) at 256 cycles down to 90 dB in one ear and 100 dB (British) in the other ear, and no response to higher frequencies She had got a lot of words nevertheless and was actually putting some of them together When I did the audiogram I turned it full on which nobody had done before and she said It s noise

(Editor s Note This is a good example of the way in which Dr Whetnall uses the term deaf Most members of the conference would probably have used the term hard of hearing in this case )

GLORIG What about the children who have not developed speech and who have this kind of hearing loss? Have you seen such a situation?

WHETNALL Yes The second child that I mentioned suffered from con genital deafness due to maternal beri beri

GLORIG Did they give you back normal speech after your type of training?

WHETNALL Yes

GLORIG In spite of the fact that they do not hear the frequencies above 1000 cps?

WHETNALL Yes

LIVC I have found the same sort of thing that Dr Whetnall reports I was faced at one point with children aged six to seven years with no previous experience of hearing and with losses comparable to the ones mentioned by Dr Whetnall One apparently had no hearing over 1000 cps In these cases I was able to get the children to produce very natural speech that is very natural intonation One of them could actually sing in tune

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## *Discussion of Benefits of Amplified Sound*

Griffiths We have a program in two sections, one is for infants, the other for pre-school children The break in age is at nine months, i e, all "infants" are of eight months and younger, pre-school "children" are nine months and older

We have had 44 infants between the ages of 30 days and eight months who have been fitted binaurally with aids These infants were diagnosed by pediatricians and otologists as having hearing losses and not responding to sound They were sent to us and we equipped them with hearing aids The infants were given formal auditory training on an individual, once a week basis Of this group of 44 infants, 33 used hearing aids for an average period of four months, at which time they were found to be normally responsive to sound They are no longer using hearing aids Four children seemed to respond normally to sound after wearing aids, but regressed and are now again using aids Two of these have a history of otitis media preceding regression Seven infants never discarded aids Whether the hearing aids were effective in changing responses to sound or not, I do not know We do know that among the children of nine months of age and older who were fitted with aids, not one child has been able to discard those aids

Among our children from nine months on, we have had many who have severe losses but who have nevertheless been able to learn speech and language sufficiently to make it possible for them to go to regular schools We find that the audiograms of some of these severely deaf children originally showed no response above 1000 cps However, after therapy (meaning the use of amplified sound) they have shown responses further up in the spectrum They now have very low level responses at 6000 and 8000 cps, which they did not have originally, after they have used hearing aids We give each child two hearing aids, one for each ear, from the day they start in our program, so they are full-time users of hearing aids and auditory training accompanies it

Chairman Thank you, Dr Griffiths You have given us clinical evidence that the use of amplified sound helps to improve the status of these children

Davis I would like to comment on Dr Griffiths' remark that none of the children in her group discarded their hearing aids beyond the age of nine months There are two possible interpretations, both of them very in-

teresting The first is that it is very important to get hearing aids on these children extremely early so that they will have plenty of training before nine months and will be able to discard them This interpretation would make the first nine months an extremely critical period Presumably this period is critical for reorganization of a faulty central nervous system because I do not believe that any of us would maintain that the use of amplified sound causes any regrowth of missing sensory cells or auditory nerve fibers in the ear itself

The other interpretation is that it is very difficult to test children reliably below the age of, let us say, six months Here we have to rely on tests of very young infants so that perhaps the question is really still wide open as to whether there really was a change in their hearing that allowed the children to discard their hearing aids or whether the first test was inaccurate This doubt leads to the next point, which is that in very young children it is very difficult to measure the improvement of hearing quantitatively either by pure tone audiometry, by speech audiometry, or by psychological testing We still rely on qualitative clinical impressions of 'improvement'

W Hardy I have seen probably an aggregate of 4000 to 5000 children and watched them develop and first learn language and finally fluent speech It is more than a casual clinical impression, although I cannot document it in detail, that it is usually possible to do a pretty fair SRT (Speech Reception Threshold) and come reasonably close to their real threshold of recognition However, there is usually a time lag It is not until the child is perhaps nine years old that we can expect him to tolerate, to respond, to pay attention, and to utilize information sufficiently to be tested by a standard adult discrimination test All of this relates to the general concepts of speech recognition as well as to the familiarity of the whole thing

I suppose I have done as much pure tone audiometry as anyone in this room and I shall continue to do it, but we must always bring speech recognition into the picture because we must assist in making judgments about placements of the children and their expectancies for the future These things depend on the integrating functions of the brain as it handles acoustic information This kind of function is not measured by the pure tone audiogram

Whetnall Here are two audiograms of the right and left ears of a child whom I first saw 17 years ago At that time I was unwise and I only put one aid on her, on her right ear In the left ear she never wore a hearing aid and doesn't really like to do so now She finds it difficult to hear in that ear and the level of sound becomes unpleasant if it is elevated further She does get a better result in a binaural speech audiogram but I think it is significant that the ear in which she does not use

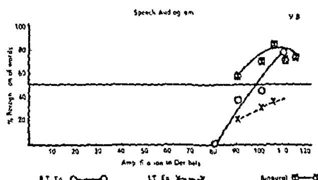



Fig. X 13 Evidence that perception improves if infant is trained to listen. The pure tone audiogram (see Fig. X 11, case 2) has not improved but the speech audiogram is better in the right than in the left ear. The hearing aid was used in the right ear. (Reproduced from *J. Laryngol.*)

the aid has not become of the same value to her as the ear in which she is accustomed to listening

CHAIRMAN So she learned to hear and understand speech on *one* side only?

WHETNALL Yes, that's right

CHAIRMAN It comes in all right through one ear and not through the other?

WHETNALL Yes

SILVERMAN Are you implying that the linguistic operation, which is quite distinct from the acoustic analyzing function, is performed better in one ear than in the other?

WHETNALL Yes

SILVERMAN Is it because more acoustic cues enter through one ear than the other or do you think that is something central?

WHETNALL I think that deafness, particularly congenital deafness is peripheral and central until the child has learned to talk and understand. It then ceases to be central and is peripheral only. It is originally a central problem of course, because

SILVERMAN (interjecting) That's what I mean! There is a peripheral acoustic operation and a central linguistic operation. I am wondering why the speech audiograms of the two ears that were originally similar became different?

WHITNALL: Yes My point is that the memories whereby we *recognize* phonemes and morphemes and our knowledge of their probabilities of occurrence and so on are stored centrally, at a level higher than the level of the sense organ That is why the speech audiograms are different This is because, as I understand it, she has never heard the sounds loud *enough* in the unaided ear for them to be of any value

SILVERMAN I am really endorsing what you are saying I am *just* surprised that the improvement takes place unilaterally I think what we need to explore further is what set of cues the child has to go by and how she uses them This kind of information will influence the kind of *auditory* training that we do I think we might do better with our auditory *training and* focus it a lot better on important points What I would still like to know is just how the child uses his limited acoustic cues

WEDENBERG For three years I gave auditory training to 12 hard of hearing children in the ages of nine to fourteen years I used complicated apparatus with both high-pass and low-pass filters With these filters I could gradually eliminate the second vowel formant or I could eliminate it abruptly

I also had a control group of children with about the same auditory impairment who never were trained Before I started the training I measured the hearing of all of the children for pure tones and also made speech audiograms for all of them

After three years I tested again and got astonishing results In the control group nothing had changed, but in the trained group some of the pupils now had better pure-tone audiograms and much better speech audiograms for vowels, words, and sentences Improvements in the pure tone audiograms were about 10 decibels at many frequencies but never more than 10 All of the pupils whose audiograms had improved had one thing in common they had no recruitment (Bekesy audiogram) On the other hand the pupils who did have recruitment showed no improvements either in the sensitivity for pure tones or in their speech audiograms

My interpretation of this was that the improvements in sensitivity for pure tones are not real, physiologically, but are due to the circumstance that the pupils had become three years older and were better listeners They did better recognizing the very faint sounds near threshold If there is no recruitment the tones are very faint for a number of decibels above threshold If there *is* recruitment on the other hand, there is a very sharp transition at threshold The threshold is sharp, irrespective of their age, and therefore the cases with recruitment show no improvement by pure-tone audiometry

SILVERMAN My point may not have got across It was that perhaps there is a bette job of analysis of the acoustic event in one ear as compared

with another, and that the difference does not depend on something linguistic happening in the brain

M HARDY I am thoroughly in agreement with most of what has just been said, but I have come across three children who present an interesting problem Their hearing aids were put on what seemed to be the better ear in each case They did not make good progress and were shifted over to the ear that was poorer by pure-tone tests They have been using their aids much more successfully and have been on them for several years I don't know the explanation of it, but I think we can "go off the deep end' by drawing conclusions from too few cases I believe that the sites of damage are going to prove more significant in determining the ability of children to discriminate than the age at which the hearing aid is put on I am a firm believer in the use of early amplification, but the type of hearing loss and its psychoacoustic consequences certainly must enter into the end result Also we must be cautious because our present tests do not reveal the whole truth to us

GLORIG I believe that much of the problem is a learning problem and that the sooner we begin to get signals into the brain the better It doesn t make much difference just what the acoustic signals are The brain will learn to do the proper job

DOWNS (Mrs Downs has here combined for emphasis some remarks that originally were scattered through the discussion )

We have had some experience with the management of what we call the Limited Hearing Child This is summarized in an article by Stewart, Pollack and Downs entitled "A unisensory program for the limited hearing child (*ASHA* 1964, *6* 151 154)

We believe firmly that, with limited-hearing children, the greatest benefits will be obtained if special measures are taken at as early an age as possible If too much time elapses before amplified sound is employed the impaired auditory sense will have less chance of becoming an efficient pathway of communication

Our goal in auditory habilitation is to develop a "listening function' in the child with limited hearing In the early stages of training of the more severely impaired child, and also for continuing training of the hard of-hearing child this function can best be developed by constant, intensive auditory stimulation De emphasis of lip-reading serves to structure the listening situation for the child We use visual experiences to direct the auditory communication but we avoid teaching of lip reading and tactile stimulation For this reason we call it "unisensory training"

A home training program in which the full cooperation of the parents is secured is needed for the best development of listening The daily life in the home should be so structured that every available opportunity is

used to give the child meaningful auditory stimulation Natural language patterns suitable to the developmental level of the child should be used with the goal of developing the normal rhythmic and tonal patterns of speech

*The concept of* hearing age is a useful one *The chronological hearing* age is dated from the time that amplification is initiated The entire auditory experience and development of the normal child is then recapitulated for the limited hearing child He must go through the same stages that the normal child goes through for the first six months he is bombarded with sound and develops awareness for the next six months he experiments with the feed back of his own voice and develops some discrimination of sounds and of gross speech after that he is ready to produce speech himself and to discriminate more complicated speech patterns It is only through such a recapitulation that a sound basis for speech and auditory discrimination can be developed

If the child has enough *residual hearing* to develop a true listening function he is more likely to be able to succeed in the normal hearing world through unisensory training than if he is trained with visual and tactile speech clues

## *Individual Hearing Aids Broad Band Hearing Aids and General Amplification of all Nursery Sounds*

CHAIRMAN Let us now consider the question of whether general amplification of all nursery sounds might take the place of putting a hearing aid on the child

(The opening part of this discussion was not well recorded but Lady Ewing and Dr Davis developed the point that both the wearable hearing aid and general amplification of nursery sounds might be valuable because they do somewhat different things The wearable hearing aid is on the child all the time but it is usually restricted in its acoustic performance Its band of amplification is narrow but general amplification can be made high fidelity and cover the whole frequency band )

SILVERMAN I think there are three points to consider The first is the value of constant exposure to sound which is attained by the constant use of the wearable hearing aid or by general amplification it doesn t matter which Secondly there is the problem of convenience and finally there i the question of whether there can be some helpful modification of the a oustic signal that might do better even than broad band amplification I thought here is that the energy might be concentrated somewhat and t by be made more useful to the abnormal ear If we learn what the rules for each ear what acoustic cues it can use and which have the most t ect we can then modify the environment or the acoustic events

that take place in it to take advantage of these rules Ideally we will want an instrument that gives constant exposure and at the same time does to the acoustic signal what we think ought to be done to it We will hope that it can be done conveniently

A EWING As you know, two principal types of hearing aid are available for use with children Of these, the first provides broad-band amplification, that is to say an amount of amplification that can be beneficial when needed, to very severely deaf children over the greatest part of the speech range of frequencies This makes possible what is described, with regard to radio and television sets, as high fidelity (i.e. broad-band) reproduction This category of hearing aid, when of the kind known as a speech training aid or an auditory training aid, is portable but not wearable Receivers are of the external dynamic type High quality microphones are fitted The group hearing aids, much used in schools for the deaf, are similar in design The other main category of hearing aids, namely those that can be worn on the person, are fitted with miniature or insert magnetic receivers which are clipped into earmoulds to be worn in the ear They provide reproduction that, for severely deaf children, corresponds in band width to that which is characteristic of ordinary line telephone systems

Figure 14 shows, in terms of frequency and intensity, high fidelity amplification provided by a master hearing aid, typical of good speech training aids and group hearing aids, as contrasted with the performance of two types of wearable hearing aids much used by very severely deaf children It will be seen that the two categories of aid, high fidelity with external receivers and wearable with insert receivers, differ greatly as regards the width of the frequency bands over which they provide high-level amplification The master aid gives much greater amplification than the wearable aids at frequencies above 2500 cycles per second

In a recent study we have compared the speech scores obtained by young partially deaf children when they are given the benefit of broad band amplification with their performance when using ordinary wearable hearing aids The pupils ranged from 7 to 12 years of age The test material was the Manchester Junior Speech material and the scores are expressed as the percentage of words correctly heard

The high fidelity amplification was provided by a master hearing aid fitted with a high-quality microphone and dynamic external earphones It is a training instrument which is portable but not wearable The audiologist spoke the words at a sound level of 70 dB within four inches of the microphone The volume setting for each of their ears was chosen by the child and the audiologist jointly, after a brief preliminary test, to obtain the most favorable setting

[illegible]ority of the children who were tested have average hearing levels [illegible] and [illegible]0 dB (Br.) With broad-band amplification 18 of the 37 children scored over 60% and 9 more, two of whom had hearing levels

*Act[illegible] to laryng 206*

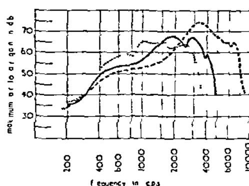



Fig. X 14 Performance in terms of frequency and intensity of a master hearing aid and two types of wearable aids used by severely deaf children The dashed line represents the master hearing aid, *the dotted line a wearable aid with insert receivers the solid* line, a high fidelity wearable aid with external receivers

over 80 dB scored between 44% and 60% Two other children with hearing levels greater than 105 dB (Br ) obtained no scores

By contrast, with wearable hearing aids, only four of all the children scored over 60% Eleven others scored between 44% and 60%

The foregoing tests were performed under quiet conditions An additional test was carried out to simulate speaking and listening in a classroom without sound treatment in an ordinary school and with a considerable noise background A tape recording, actually made in an ordinary school, was replayed while each child was being given the speech test The tests with the wearable aids were made with the child 10 feet away *from the audiologist in order, again, to simulate normal school conditions* The audiologist monitored his speech to maintain a level of 70 dB at the microphone of the children's hearing aids throughout all of the tests This loudness level was the highest recorded by Sanders in 1961 as typical of *teachers' speech in ordinary schoolrooms*

For the tests "in noise" the tape recordings were played from a loudspeaker at a mean sound level of about 60 dB measured at the child's

TABLE X 2 *Speech Reception by Master Hearing Aid and Wearable Aid in Quiet and in Noise*

Distribution of Children by Scores

| Type of Aid | Scores (percent correct) 0 | 4 20 | 21–40 | 44–60 | 64–80 | 84–100 |
|---|---|---|---|---|---|---|
| Maste | 2 | 5 | 3 | — | 13 | 5 |
| Wear aid in quiet | 1 | 10 | 11 | 11 | 3 | 1 |
| Wea aid in noise | 2 | 13 | 17 | 4 | 1 | — |

hearing aid. The resulting signal to noise ratio was more favorable to the hearing aid users than many of the ratios that were determined by Sanders in ordinary schools.

The children clearly heard better with the broad band amplification provided by speech training and and group aids than with their wearable aids, as shown in the following table.

## *Low Frequency Amplification*

CHAIRMAN: The next item on our program reads: What can be expected for children with only low tone residue of hearing? Might they be helped by frequency transposition? Mr Ling tells me that he has some pertinent experiments to describe to us but that the question should be: Might they be helped by low frequency amplification? By this I believe he means extending the pass band of hearing aids to include more low frequencies.

## *Reproduction of Frequencies below 300 cps for Children with Residual Hearing*

This paper is based on material to be submitted in partial satisfaction of the Degree of M.Sc. in Research of McGill University. Thanks are due to Dr D. G. Doehring, Dr B. P. Cannon, Professor A. Rigault and to Mr J. Frydman of McGill University for their help and to the Zenith Hearing Aid Sales Corporation, Chicago, for the provision of these aids designed to the writer's specifications.

LING: Children who have only a low tone residue of hearing constitute a significant proportion of cases enrolled in schools for the deaf. As the range of frequencies to which these children can respond is extremely limited, standard hearing aids, which have a frequency range of approximately 300–3000 cps, are of little value. Fitted with a hearing aid having a lower limit of 300 cps a child with no measurable hearing above 500 cps has less than an octave bandwidth available to him. This bandwidth carries too little information for a child to learn through hearing. Hearing aids of the standard type merely expend their energy over several octaves to which children with only low tone residue are completely deaf, however powerful such aids may be.

Because fundamental voice patterns and the lower formants of speech for both male and female speakers occur within the frequency range available to children with residual hearing, the writer experimented with a hearing aid designed to reproduce frequencies as low as the fundamental of a male (bass) voice in comparison with a standard hearing aid with the response characteristics shown in Figure 1a.

Comparative spectrograms of the performance of the two instruments in producing speech were made by the following procedure. A standard [illegible] of the writer's left ear was obtained and additionally drilled to [illegible] inch plastic tube with an internal diameter of 2 millimeters

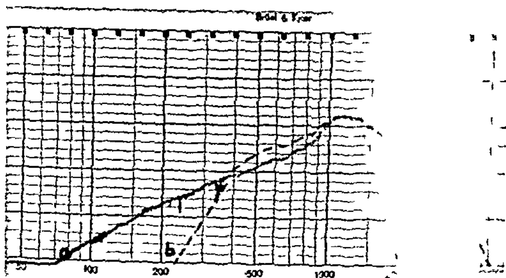



FIG V 15 Air to air characteristics of (a) experimental hearing aid (b) ... aring aid

leading to a probe microphone so that the sound actually presented to the eardrum could be analyzed The sentence Father gave me shoes was recorded on a Crown Model 1000 full track tape recorder at a speed of 15 inches per second by both the writer and a female speaker The sentences were then played back through an Ampex Model 2010 amplifier and speaker into a sound proofed room where the writer wearing the earmold was seated The speech sample presented under the following conditions was recorder from the writer s meatus on the drum of a Kay Sona Graph Model M recorder/analyzer via a Bruel & Kjaer probe microphone and amplifier type 2603 for both male and female speakers

(a) into the open earmold at a distance of one yard from the amplifier speaker
(b) through the experimental hearing aid with its microphone one yard from the speaker and the receiver connected to the earmold
(c) through the standard hearing aid with the microphone at the same distance from the speaker the gain adjusted to the same point and the same receiver as (b) above connected to the earmold

Comparison of spectrograms taken under conditions (a) and by direct recording onto the Sona Graph drum showed that the 9 inch tube leading to the probe microphone acted as an acoustic filter for high frequencies but that no distortion of low frequencies was introduced by the method employed For the purpose of this study, normal spectrograms wide or narrow band over the normal ranges 85–4000 cps or 85–8000 cps would have been too insensitive to provide adequate information about the low frequency components of speech The Kay Spectro-Graph was therefore

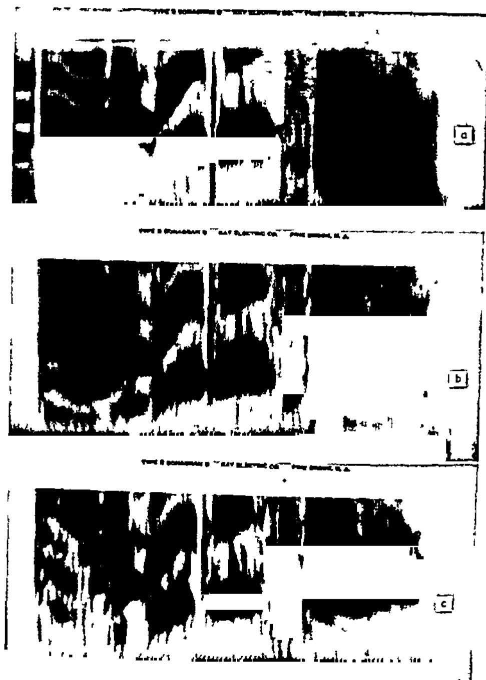



Fig. V 16 Expanded and magnified narrow band spectrograms showing low frequency components (0 500 cps) of the sentence "Father gave me shoes" spoken by a male and recorded by means of a probe microphone from the listener's meatus top (*a*) through open earmold centre (*b*) through experimental hearing aid and bottom (*c*) through standard hearing aid

modified so that the frequency range 0 500 cps could be studied in detail Expanded and magnified narrow band spectrograms with this frequency range were obtained as shown in Figs 16 and 17

Fig 16 *a* shows the pattern recorded through the open earmold The




FIG V 17 Expanded and magnified narrow band spectrograms showing low frequency components (0–500 cps) of the sentence "Father gave me shoes" spoken by a female and recorded by means of a probe microphone from the listener's meatus (*a*) above, through the experimental hearing aid and (*b*) below, through the standard hearing aid

calibration signal, at intervals of 100 cps, is shown on the extreme left of this spectrogram Fig 16 *b*, the pattern recorded through the experimental hearing aid, shows the male fundamental to be slightly attenuated below 100 cps but otherwise identical to the open earmold condition (Fig 16 *a*) Fig 16 *c* shows a severe attenuation and distortion to the pattern to result from condition (*c*) in which the speech sample was transmitted through a standard hearing aid

Fig 17 *a* shows the pattern of a female voice recorded via the experimental aid, which is identical to the pattern obtained through the open earmold Fig 17 *b* shows the pattern recorded through the standard hearing aid Comparison of the two spectrograms in Fig 15 indicates that while the experimental aid is perfectly satisfactory for the reproduction of the low frequency components of the female voice, the standard aid attenuates the fundamental, in the middle of the sentence, which drops to approximately 250 cps

Preliminary observations of the responses of children with low-tone residual hearing using the experimental, as opposed to standard, hearing aids indicate significant improvements in the perception and production of speech

The primary advantage of the experimental instrument is that it widens the auditory field of the child with residual hearing Improved audibility of speech sounds having low-frequency formants leads to an almost immediate increase in the quantity and quality of spontaneous babble among babies and to greatly improved imitation of rhythm and stress patterns in words and phrases among deaf children who have acquired some speech formerly through standard type hearing aids Experiments using standard hearing aids have shown that, to children with residual hearing, a sentence such as "We have two ears" may be perceived as one syllable at 15 feet, as two syllables at 10 feet, and as four syllables at 2 to 3 feet It is not surprising that the stress and rhythm patterns of deaf children's speech is generally so poor when such unstable patterns are available to them The experimental hearing aid, by approximately doubling the distance at which phonemes with low frequency formants (such as /u/ and /i/) are audible to these children, does much to remedy this situation The improved low-frequency response of the experimental aid also helps to provide more stable patterns of pitch and intonation, which, like speech rhythm, can vary with distance for children with only a low tone residue

In summary, let me repeat that ordinary hearing losses extend widely over the frequency range so that there are usually plenty of auditory cues available in the high and middle frequency for pitch, rhythm, intensity, and so on The harmonics as well as the fundamentals of the voice are heard In this situation we don't need the fundamental, as we know from the telephone The children with residual hearing, however, do not hear these higher frequencies, but can hear the fundamental and the first few harmonics With the fundamental alone they can detect and learn to reproduce the correct pitch pattern, whether it is a rising or a falling inflection It is the lack of this inflection, taking no advantage of pitch cues that *tends to produce the monotonous voice, without stress, without movement,* without intonation, that is so common amng children in schools for the deaf By taking advantage of their low-frequency hearing residue I have been able to get severely deaf children to reproduce pitch, intonation pattern, stress, and rhythm very much more readily

Another great advantage is that with the added emphasis that such a hearing aid provides in the low frequencies, the young child's field of audition is greatly extended Instead of hearing only a few sounds at a short distance, a baby will be able to hear all voiced sounds a great deal farther away This means that his auditory attention can be focused on sound with the result that speech can become meaningful to him vary with distance for children with only a low-tone residue

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CHAIRMAN Am I correct that, with the severely handicapped children it is not a matter of increasing the intelligibility of the speech that they hear but rather its musical and rhythmic pattern? This in turn improves their speech patterns?

LING That's right, but I think that this frequency characteristic can also be useful for children who have more than just residual hearing because we have found that it helps them to discriminate voice stress Another point is that the younger the child, the greater his response to this sort of thing We are looking for the basis of this low-tone hearing We believe that sometimes it can be true hearing sometimes merely feeling vibration, sometimes both This low-tone hearing is not a simple matter

DAVIS I agree that there is a large component of tactile stimulation in this low tone residue This I mentioned when I pointed out that one of the things that we are working on with our cortical evoked responses is to try to separate the tactile from the auditory stimulation in exactly this type of case

A EWING I believe that further research on selective amplification for severely hard of hearing children should be very valuable

DAVIS (an afterthought) I agree again, and I would point also to the children with so-called "hammock audiograms" which rise toward both ends of the frequency spectrum If their low frequency hearing is actually only tactile sensation they may not get much real help from it but they might benefit from an extension of the *high-frequency* range of their hearing aids In any studies of selective amplification I am sure that the presence, the location and the extent of any "island" of true hearing in each child will prove to be of critical importance

WEDENBERG In auditory training I depend upon the formants of the speech sounds When a child has lost his hearing above 1000 c/s, I must work out a special program for him

One must begin very early, with the *live voice*, close to the ear if necessary, to teach the child to differentiate between vowel sounds such as oo and "aw" The first formant of these two sounds is around 250 c/s

This gives the child an impression of the vowel sounds even though he *can hear only the fundamental and the first formant* Then I *add the* sustained voiced consonants "l", "m", "n", "r", of which the child also *can hear only the first formants. I next compose single words, using only* these sounds whose first formants the child can hear (ya; mama) Then *I proceed to two-word sentences (ya, mama)*

Even though the child is not able to hear above 1000 c/s he is able to recognize *fricative and plosive consonants by cutaneous sense, and can be* taught to perceive the whole language.

When a child has been trained thus by live voice, if he uses a usual hearing aid or a hearing aid with extended range in the low frequencies it is not possible for this child to hear the unvoiced consonants and therefore very often the child actually approaches the instructor asking him to speak into the unaided ear A coding amplifier, the so-called transposer designed by Mr Bertil Johansson, is of great value by overcoming many of these difficulties With the help of the transposer the child after training is able to a certain extent to discriminate the transposed fricative sounds with the hearing sense

### *Hearing Aids that Use Frequency Transposition*

CHAIRMAN I had thought that Mr Ling was going to tell us about hearing aids that use frequency transposition By this I mean a systematic moving down of high frequencies into the lower frequency range so that a pattern corresponding to the speech pattern is brought within the competence of children who have only a low-tone residue The hope is that the pattern can then be analyzed and learned, essentially as a new form of language This kind of thing is done by Dr Pimonow in Paris using one system and Dr Johansson in Sweden who is making a different engineering approach It is an interesting question whether our auditory discrimination in the low frequencies is good enough to allow us to recognize enough difference to make speech intelligible The speech certainly sounds *different*!

WEDENBERG Many of the severely hard of hearing have hearing *defects* of the perceptive high tone loss type with no hearing above 1500 c s The greatest difficulty in the use of hearing aids by pupils in *this group lies* in the amplification by the hearing aid of excessively low frequencies where *the hearing loss is less than in the higher frequency range Overloading,* causing masking of the higher frequency range which is more valuable to *understanding, occurs especially with high amplification, and the threshold* of discomfort is easily reached The information obtained from the lower *frequency area is also limited in comparison with that obtainable from* the higher frequency range The unvoiced consonants, which are so extra*ordinarily important for the discrimination of speech, are* not perceived at all because the most important unvoiced consonant formants lie above

Fig. V 18 Pure tone audiogram with a threshold curve and formants from a series of Swedish speech sounds (according to Fant) Three voiceless consonats are indicated after transposition

3000 c/s, and the hard of hearing often have no useful hearing above 1500 c/s Connected speech is therefore impossible to discriminate except with a combination of hearing and lip-reading In order to try to overcome some of these difficulties Mr Bertil Johansson built the coding amplifier or the transposer

With the help of a 4800 c/s carrier frequency he achieves an intermodulation between this carrier frequency and the frequencies constituting the main part of the unvoiced consonant formants, resulting in a transposition of the 3000–6000 c/s frequency range into the range below 1500 c/s where the hard of hearing have their residual hearing Through the transposition of the frequency range 3000–6000 c/s (=3000 c/s) into the range 0–1500 c/s (1500 c/s) a frequency compression is also brought about The transposer also provides amplitude compression

The impressions of the hard of hearing of the transposed fricative or unvoiced sounds differ, of course, from those of listeners with normal hearing The hard of hearing describe the transposed fricative *sounds* as different scratch sounds, which can nevertheless be discriminated to a certain extent from each other This means that the hard of hearing receive a new hearing pattern, which takes some time to acquire The changed ditory pattern also embodies distortions of the sounds, especially the

vowels, depending upon the superimposition of the simultaneously transposed third and fourth vowel formants

For auditory training with the transposer I selected six pupils aged from 10 to 29 years. The period of auditory training covered two months, half an hour four days per week, sixteen hours total. One of the pupils had also used a portable transposer for a period of one year. Before the training began, the hearing of the pupils was carefully mapped.

The training was carried out as follows. After the transposer with a 4800 c/s carrier frequency had been adjusted to the pupil's most comfortable level, he was trained in perceiving different consonant combinations, spondees and, gradually, connected speech. There seemed to be great individual differences in the capacity for acquiring the new auditory pattern. The material is too limited, of course, to allow the drawing of general conclusions, but it appears that early methodical auditory training is of great importance if good results are to be obtained with the transposer.

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## G. POSSIBILITY OF INJURY BY AMPLIFIED SOUND

CHAIRMAN: There have been one or two allusions to the possibility of *injury to the hearing of infants by incautious exposure to very loud sound*. There are two situations in which such danger might occur. One is the case of a child who is actually hard of hearing from some sensori-neural disorder and whose hearing loss might be increased by overloading the ear with sound. The second situation is the case of the child whose ears are normal or nearly normal but who, because of some central condition, does [illegible] speech and is considered to be deaf and is therefore mistakenly [illegible] plified sound. Dr. Glorig will open the discussion of these points.

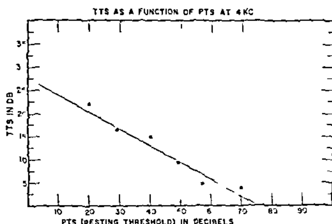



Fig X 19

Glorig Whenever the use of powerful hearing aids for suspected severely deafened young children is discussed, the question of whether further loss is produced by high level signals must be considered

The output of high gain hearing aids is unquestionably enough to produce a hearing loss in normal or near normal ears if they are worn consistently with high gain outputs

Extensive research has shown that the production of noise-induced hearing loss is inversely proportionate to the level of the resting threshold, i e, as the resting or permanent threshold level increases, the amount of noise-exposure necessary to produce a further elevation of hearing level increases When a maximum permanent hearing level is reached at approximately 75 dB, further increase of hearing level is very unusual, particularly in frequencies below 2000 cps Figure 19 demonstrates this phenomenon very well, since it has been shown that if no TTS (temporary threshold shift) occurs, no change in resting threshold is produced

Because of the difficulties involved in testing infants, there is always the possibility that a child said to be deaf actually has normal, or near normal hearing In such cases, what happens if a high gain hearing aid is used? In my experience, a young child will not tolerate sounds that are above maximum comfort level long enough to do any damage, without giving some easily recognizable response which clearly indicates his discomfort

## *Reference*

Damage Risk Criteria and Noise Induced Hearing Loss *Archives of Otolaryngology* October 1961 Vol 74 pp 413–423

Chairman Do you think, Dr Glorig, that even if the child doesn't interpret the sounds, the noise can still hurt him if it's loud enough? Will the normal protective reflexes still operate? Will the aphasic child still feel pain?

GLORIG I don't care whether the child interprets the sound or not If the energy is there it will hurt him

J HARDY Is the reflex protective mechanism effective in young infants who can't pull an instrument off?

GRIFFITHS When we expose normally hearing infants to amplified sounds they respond within about 30 seconds by crying The sound does not have to be very loud You barely turn on the volume of the hearing aid and the child's face begins to change and he begins to cry There is an almost instantaneous uncomfortable look of distress and then the response

DAVIS Do you then turn the gain down or do you leave it on for his own good?

GRIFFITHS With the normally hearing infant we don't leave it on at all

DAVIS But if a child has been diagnosed as not hearing normally, when he begins to cry, what then?

GRIFFITHS The child who is not hearing normally usually doesn't cry when the volume is turned up He has a different look entirely He tolerates it and not only tolerates it but responds with pleasure We do not put loud hearing aids on babies We have nothing that goes above 50 decibels output and the babies are usually not disturbed at this level

DAVIS Do you mean 50 decibels output or 50 decibels gain?

GRIFFITHS I'm sorry I mean 50 decibels gain

DAVIS What might the maximum acoustic output be? Perhaps 100 dB sound pressure level?

GRIFFITHS Perhaps 110 dB

STATTEN It seems to me that this would be a very good diagnostic test We are looking for ways of finding deafness in young children Can't we just put a hearing aid on and watch for the response?

DAVIS Well, I must say that this reminds me of some of the tests that we have already seen illustrated in which loud sounds are delivered very close to the infant's ear

GRIFFITHS May I just say that if a doctor sends a baby to us and says that the baby has a hearing loss and then we test it and it seems not to

hear we then try an aid on it If the baby cries we say that the test is wrong and the baby is right and we do not put an aid on For the baby that it is difficult to ascertain whether a hearing loss exists or not a series of re-evaluations are scheduled in four to six weeks intervals in order that further studies can be made

DAVIS In other words you substantiate what Dr Glorig says Even if a baby does not tear the hearing aid off he gives a sufficient sign or a kind of cry that you recognize and you act accordingly Very good

W HARDY This is one of the questions in our field that has suffered from too many anecdotes and too little experimental work Actually the work that Dr Glorig just presented couldn t have been talked about a few years ago but I know of no really good controlled research that deals with the reactions of young children in this situation I have been preaching the early use of hearing aids for many years now but I still hold to the perhaps old fashioned belief that we really ought to know what our tools are for relative to the problems of the child we are discussing This had ramifications in terms of the so-called aphasic child

We have used many mild gain hearing aids on children who are of course beyond early infancy in whom we suspect disorders of sensory integration We used mostly one particular hearing aid with about 35 decibels gain and a maximum output between 85 and 90 dB sound pressure level and Dr Statten Talk about a diagnostic tool Many of these children at $2^1/_4$ and 3 years of age seem to get more information this way They seemed to have basic disorders of sensory integration with respect to language memory, and recall but by the age of 5 or 6 years they d n t want the hearing aids any more

The hearing aids were not given to them with the expectation of remedying the peripheral hearing impairment but as a potential help to assist them in sensory integration but I would feel better if I knew a little more about what is wrong with a young infant before moving too far too fast

LING In the course of our work in Reading England we noticed that many children who were using hearing aids over a period of years showed some deterioration of hearing something like what Dr Harr has described We wondered if the deterioration could possibly be due to the use of hearing aids We set up an experiment to test the production of auditory fatigue in these children We measured the auditory fatigue by a technique of binaural loudness balance In some of the children we could demonstrate a small amount of auditory fatigue after several minutes of exposure to loud speech sounds while in others we could demonstrate no fatigue at all

I am glad to say that most of the deteriorations that we observed only amounted to a few decibels on the average but we were unable to determine with certainty whether or not the hearing aids might be implicated

RØJSKJÆR (afterthought). It seems to me that the best way to avoid any possibility of injury to hearing by amplified sound is by careful diagnosis in the first instance and, if in doubt, by limitation of noise exposure by adjusting the intensity or the time schedule or both

I read a paper on that subject at the Fifth International Congress of Audiology in Bonn in 1960 Three hundred and ninety cases, both adults and children, with different kinds of hearing diseases of the inner ear and treated by hearing aids for five years or more, were reexamined We found 9 who since the first hearing examination had developed additional hearing loss in the ear using the hearing aid This ear alone was worse than before, the other ear was unchanged It might be a series of coincidences, and we cannot exclude the possibility that it might be a progression of the original disease in the hearing-aid ear only Against the chance of coincidence is the fact that no cases were found with the opposite findings, that is further impairment of hearing in the ear without the hearing aid Our patients use their hearing aids all day and every day

In all procedures of treatment it is important to examine both positive and negative results with respect to the future, including the risks from the treatment itself, but this risk must not restrain us from treating our hard of-hearing patients, as well as our hard-of-hearing children with little residual hearing, with hearing aids The hearing aid is of such great importance for the acquisition of language and speech and for education as a whole that we ought not to ignore it, since the residual hearing in the children is of no use if sounds cannot be amplified We hope that further development of audiology will assist in the elimination of the risk of acoustic trauma by amplified sound, particularly by identifying in advance those patients especially sensitive to injury by noise Developing special hearing aids should also help

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## H STABILITY OF PURE-TONE AUDIOGRAMS

CHAIRMAN Dr Barr, I believe you have some comments on the stability of pure tone audiograms in children

BARR At Karolinska Hospital, 149 children and young people with severe hearing impairment of known aetiology have been re-examined by Dr We-

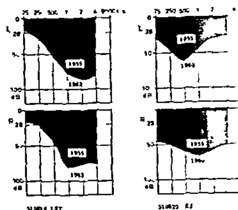


FIG V 20 Follow up examinations in children with exogenous pairments Typical audiograms in cases with hearing loss and a paranatal anoxemia (LST) and in cases with hearing loss ca (EJ), none of them showing any further loss during time of Hearing loss at first examination and follow up L=left, R=righ

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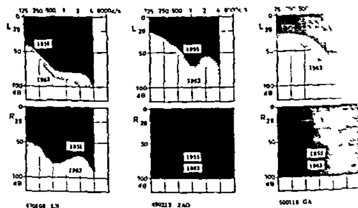


FIG V 21 Follow up examinations in children with exogenous (acquired) hearing impairments Audiograms from three children with hearing loss caused by meningitis and or dihydrostreptomycin Black area hearing loss at first examination Dotted area additional loss since first examination L=left R=right ear

denberg and myself up to 15 years after the first reliable audiogram was obtained by either octave or Bekesy audiometry The majority of the children have been using hearing aids regularly and many of them have been subjected to intensive auditory training Children belonging to the "maternal rubella" and the "acquired paranatal" groups (anoxia, birth injuries cerebral palsy, prematurity (less than 1500 g birth weight) or kernicterus showed neither progress or regression of their hearing thresholds A larger percentage of those with hearing defects following meningitis and or di

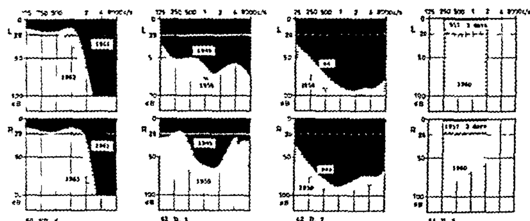


FIG V 22 Follow up examinations in children with endogenous (hereditary) hearing impairments Black area hearing loss at first examination Dotted area increase of hearing loss since first examination l = left R = right ear (Barr, B., and Wedenberg E., 1965 Prognosis of perceptive hearing loss in children with respect to genesis and use of hearing aid *Acta otolaryng* (Stockh ) 59 462 )

hydrostreptomycin treatment and endogenous (hereditary) hearing defects showed obvious worsening of hearing *The results speak against any possible beneficial effects of amplified sound measurable by pure tone audiometry*

DAVIS Perhaps the point here is that the pure tone audiogram never improves in these older children and in some cases it actually seems to deteriorate What the deterioration may have to do, if anything with the use of a hearing aid or amplified sound, is not at all clear Personally, I do not expect the use of amplified sound to improve peripheral hearing The sensitivity of peripheral hearing is what we measure by the pure tone audiogram I think of it as something that is anatomically and physiologically quite stable except perhaps in such situations as the congenital progressive deterioration mentioned by Dr Barr and Dr Wedenberg The results of speech audiometry are not so stable Speech audiometry cannot be done in the first place until the child has learned some speech to use as material A central functional impairment may be quite radically modified by the use of amplified sound Learning may take place and allow the child to make full use of whatever physiological faculties he may have In Dr Whetnall s sense the child becomes ' less deaf"

RAPIN I am interested in developing new techniques for audiometry in children and I require a reliable measure against which to validate them At present I use the audiograms, and I also look at the diagnoses The diagnoses are often so uncertain and in such a state of flux that they are not very useful for us in terms of validation The audiograms despite their frequent unreliability are still the best criteria we have May I make a plea for good and reliable audiometry

# VI. DEVELOPMENT OF LANGUAGE AND THE "CRITICAL PERIOD"

CHAIRMAN. I ask Dr Tervoort to open the discussion of the development of language in young children I understand that some of his remarks will deal with the development of esoteric language in children who may or may not be deaf and that he will have some comments also specifically on the deaf child

TERVOORT· The normal hearing child masters his mother's language within the first three years of life After one month the babbling, that is voice-giving with movements of the future speech apparatus, begins to differentiate, after six months the babbling starts to adapt itself to the phonemic pattern of the language around, after twelve months the first one-word sentence occurs, i e the first symbolic reference with the same sound group for the same reality in different situations is used, after eighteen months the child begins to combine words in syntactical patterns, and after 24 months he begins to use the more intricate compound structures as well as the correct morphological pre- and suffixing, both of which are thoroughly trained around the completion of the fourth year of life The system of language thus mastered is a very complicated system of symbolic references, as well as a very effective system of communication Anyone can discover this suddenly when listening to the fast and faultless talking of a three-year old child in a language he is trying to master himself Through this system of symbolic references of communication the child learns to control his environment, to understand his world, to communicate with his fellow human beings in a very fast and effective way. We might correctly say that mastering the language is tantamount to the mastering of life itself

The ear plays a decisive role in this learning process My research in abnormal, esoteric developments has to be seen against this normal development Language understanding basically is distinctive sound perception, and fundamental features of this sound perception are its distinctive audibility, its linearity, its arbitrariness of the symbols used, based upon agreement of the communicating partners, and finally its multidimensional characteristics, such as the possibility of word combinations, multiple meanings, metaphoric usage, and the like As a consequence not only the specific sense organ but also the sensitive age for learning the language plays a decisive role There are indications that there is a direct correlation between

the age at which a language is learned, and the success with which this is accomplished. There is evidence that the optimal age for learning the mother tongue lies before the pre-school age; that delay for all kinds of reasons causes retardation, and that complete success is impossible after the age of pre-puberty is reached. From my own personal experience I am convinced that this is true, and that the notion of the sensitive age is an extremely important one in the learning process of language acquisition. However, I would welcome other, such as neurological, evidence as to the amount that a delay of this learning process—for the purpose of first establishing an extensive vocabulary through finger spelling and reading only, for example—would do permanent damage to its successful realization.

There is no need to work out the absolutely fatal consequences of congenital or prelingual deafness to the development of language for the learned members of this convention. I just want to renew your awareness of these facts in the following sequence: first, a brief summary of facts that do not happen in the life of the very young deaf child, secondly, the wrong course this life usually takes, and third, the course it should take and actually does take in a minority of cases, thanks to pioneers in the field.

First: things that do not happen, there is sound giving at the beginning but there is no proper self perception or perception of others and therefore no cybernetically controlled feedback training and differentiation of the future organ of speech. On the contrary, there occurs a flattening and a dulling of the natural voice quality, and even a process of muting. The latter seems to occur more frequently as one of the traumatic aftermaths of the fatal illness which caused the deafness, and less often in congenital cases.

Now I would like to say something about the deaf child at this early age that is not given proper consideration, and finally, about the child that is treated properly. If the deaf child is left by himself there is no adaptation to the world around of the speech sounds, and therefore no extensive training of all possible phonemic combinations prior to their symbolic usage as words. Consequently there is no single word phase, no morphological and syntactical refinement, in short, no language learning. And therefore and consequently there is no subtle and effective control of the environment, no highly sophisticated system of symbolic references, and no totally satisfactory interhuman emotional communication. I stress the fact of the pathological situation both on the symbolic referential as well as on the social communicative level because the consequences of these two reveal themselves in different areas.

What usually develops instead, apart from the negative, atrophical process of decreasing voice capacity, is the origin and the organization of an esoteric and visual system of communication, the extensive description of which I have given elsewhere. Where distinctive audibility is the fundamental prerequisite for acoustic linguistic understanding and for the oppositional

qualities of our language on the phonemic, morphemic, and syntactical level, distinctive visibility sets the norms for a communicative symbol-system controlled by the eye One of the immediate consequences is that there is no linear auditive succession of utterances, and therefore no necessary grammaticalness of the system Instead, there is the possibility of simultaneity of the symbols, as both hands, the head, the face, etc, can convey different messages at once, which can be perceived by the eyes either simultaneously or at least without strictly formalized, systematized succession I am talking about a primary and esoteric system which develops between the deaf baby and his untrained mother, or between deaf pre-school children in a tolerant and old fashioned kindergarten situation This way, the deaf baby of less than 24 months of age arrives at his first symbolic recognitions and references by means of sublinguistic, imitative or pointing gestures These gestures are motivated insofar as they resemble the things or realities meant, in opposition to the words of normal language which are arbitrary symbols, based upon agreement The latter characteristic is the prerequisite of abstract, multidimensional, metaphoric, etc, usage, all of which is lacking in the esoteric visual system The word follows the creative thinking and stimulates it, the visual sign prevents it from developing Visual signs formalize very soon, however, into agreed upon symbols, the meaning of which is evident to communicative partners only When the deaf baby has reached the age of three he has fixedly settled in a system that is utterly foreign to the symbolic communication system of the acoustical world around him

Even a preschool that takes him when he is hardly four years old, and that tries to superimpose an oral system on him, is far too late The deaf child has learned the esoteric instead of the normal system, in the sensitive years, moreover he lacks the specific organ for learning this normal system fast and efficiently at this later age The two philosophies, opposing each other educationally, are to let his so called natural manualism have its way more or less to prevent frustration and to promote learning, or, to still adapt him to the acoustically oriented world around, in order to integrate him as an adult in our world Both are compromises, and only partially successful

In my third point I would like to indicate a solution which has proven its effectiveness already, and which in my opinion is the most important step forward in the field of the education of the deaf, together with the improved electronic acoustic equipment and its multivaried usage Through the pioneering work of the late Lady and Sir Alexander Ewing Mrs Tracy and those who were inspired by them we are reaching the baby at the earlier age now, where we teach him through the mother and bring him to face consciousness, and to emotional understanding of face-to-face mimicry at first, and to beginning symbolic understanding of lip movements, and later, to voice play, babbling, and the beginnings of speech

I feel that the following steps could be made at the earliest ages

1) As far as vision is concerned, establish face to face contact in an emotionally highly significant person-to person relationship, educate the awareness of the symbolic meaning of the lip movements in the emotional context of mimicry

2) As far as sound is concerned, keep the natural voice quality by encouraging the voice-giving, specifically the babbling, educate to the beginning of real speech And all this should be done at the earliest age

CHAIRMAN Dr Tervoort, would you kindly repeat the ages at which the successive stages of normal development of speech occur?

TERVOORT I make these statements in terms of six-month stages because it is easier to remember them that way Babbling begins after three to six weeks, and by six months the baby has trained his future speech organ After six months he begins to adapt to the world around him

DAVIS In this early stage is there evidence from the behavior of the youngster that some kind of auditory feedback is involved? This might throw some light on some of our rather vague physiological speculations as to when the presence or absence of such auditory feedback might make a difference in the development of speech

TERVOORT I have made recordings of babies of eight, nine and ten months of age Dutch babies, English babies and one Chinese baby who had not been exposed to any language other than Chinese I let my students listen to a baby and from the babble they can recognize whether the baby has been exposed to their own language or to a "foreign" language They cannot recognize the baby as foreign if she is only four or five months old but by nine months of age a Dutch baby sounds foreign to English people It is beginning to sound Dutch

DAVIS In other words some learning is taking place by this time in the way of patterning the audio vocal system The inference is that it would pay to begin auditory training in hard-of-hearing children by this time

Do you have any opinion, Doctor Tervoort, as to how late this exposure to sound with its establishment of auditory feedback might be deferred without making any difference in the long run?

TERVOORT You mean in deaf babies?

DAVIS Yes, I am looking for an argument for beginning auditory training before a certain age

TERVOORT I believe that the best results can be obtained by an early start and I think that certainly the home training program should be

begun by the end of the first year I have to be careful because there is so little statistical data available as yet, but this is my intuition

DAVIS I agree that the question of the critical age for starting auditory training is a matter of intuition for each of us at the present time I think that our estimates may vary over a range of six months or so, but I personally feel that the information that we have exchanged today points clearly to the desirability of beginning not later than one year of age and perhaps earlier

TERVOORT May I point out that when a baby of six months of age produces speech sounds that are characteristic of his particular language, it means that he has been listening and adapting himself passively to these speech sounds for several weeks if not to say months First he listens, then he tries to imitate them, although incorrectly, and finally arrives at a correct imitation The beginning of this active adaptation is about six months of age

DAVIS Perhaps we are beginning to extrapolate as to when passive exposure begins to be significant

## *The Question of a "Critical Period"*

DERBYSHIRE I understand, from studies of the language of birds, that when a bird has listened to its mother it can be isolated for months in silence but later will practice the sounds and will sing exactly like its mother It seems to have this peculiar ability of being able to listen and remember at a time when it cannot imitate

DAVIS Perhaps this ability is associated with the phenomenon of "imprinting" in birds, which is far stronger than it is in humans, if indeed it exists in the human I think we must be a little careful about carrying over conclusions from the bird, which has practically no neocortex, to the human, which has a highly developed one

SCOTT The Eastern and the Western Meadowlarks have different songs but if you expose an Eastern Meadowlark to a Western, it will pick up the Western song

HUIZING Individual hearing aids give children a better chance to establish feedback Parents often tell us spontaneously that the child makes less noise when his hearing aid is on This seems to be some sort of a feed back

DAVIS I believe that the feedback is an important advantage of the individual hearing aid as compared with the overall amplification of other people's sounds The feedback of the child's own voice is clearer, stronger and more direct

LOWELL It will be helpful for us to distinguish between two different usages of the term "critical period" One is that a child must be of a certain age before he can handle certain types of tasks Another is that if a learning experience does not take place at a certain time it will be more difficult to master it later I think sometimes these are confused, particularly in thinking about the deaf child, and in evaluating the possible consequences of early deprivation

CHAIRMAN. In the outline for our discussion the point was supposed to be, 'How long can we wait, before initiating the use of amplified sound, without developing later serious problems of learning or social adjustment?" Some of us have said it best to start at six months Others might say we could wait until 18 months without making very much difference although the result might not be quite so good I believe this question is still wide open and that we don't have enough actual observations to answer it We need more information. This is a major problem for the future

W HARDY In this context I would like to ask the otologists and audiologists here who see children clinically what is the average or mean age for first visits Some years ago I examined our records and found that it was 2 and 7/10 years of age Nowadays it is about 2 and 2/10 years I think there is a real lag here and that we should do a better job of publicizing to everyone, not just to specialists who already know about it, the need for early beginning of special handling of the hard of hearing or deaf child

WHETNALL. I have had considerable experience that shows that the improvement is very great if you put a hearing aid on a child before the age of six months The child starts to babble, and if an infant is left without an aid until it is nine months old its babbling falls off and the child's progress is retarded The child who reaches the age of a year or 14 months without an aid is beginning to be inhibited with respect to sound It is more difficult to test such a child in its second year of life, especially with sounds that have no meaning It begins to take no notice of sound

Without early experience with sound it will take longer to get the child to like sounds again and much longer before the infant starts to babble and use its own voice After the age of two years the problems become still greater The child falls two years behind a normal hearing child in the matter of listening and we can't expect him to develop speech for

another two years after this time It is true that th begin producing short sentences but not fully developed la l comprehension of speech adequate for going to school l dren go to school at five years or even four years of age ar who drop behind in the educational system create a prob up to the age of five one can still train these children l as occurred that the diagnosis was not made earlier l he mother too much by saying that there is no longer any y that the child must have individual attention I have a h e these children now at the ages of five six, and seven w l ve been missed We find that they do respond to sound b ike three years at least for them to learn to talk All of th ven individually but it is a costly method It is really much ch nd effective to get them at a much younger age

CHAIRMAN I think a special point here is that it is easi 1 aid on early and prevent later difficulty than it is to go thr n us training later on We mustn t rely too much on what *can* l ater by special education but rather prevent the greater difficulty by rly treatment

Dr Brill I believe you have some comments on the matter f training and language

BRILL I am one of the few people here who has not worked in a clinic but I have responsibility for educating this sort of child in large numbers We must accept them because there is no other place for them to go Of course this situation is not unique for us Various state schools in the United States and provincial schools in Canada all must serve broad geographical areas

Dr Hardy talked about a lowering of the age at which children first come to clinics, but I am sure that he agrees that there are many many children who *never go to a clinic at all* before they are ready to start school and then they simply start school

I don t think anybody would say that there should be a deliberate delay in starting the use of amplified sound or in an educational program I would agree that when there is a delay it is going to be very difficult if not impossible to make it up

Many of us have to face the fact that a huge majority of handicapped children are not going to be in ideal situations Dr Miriam Hardy stress l the need to educate parents in broad groups There has been some m v ment in this direction in several states notably Illinois, Michigan N w Mexico and now California We are beginning to do something for e children at the preschool level when they are still not old enough acc l ing to state laws to attend school We begin with parent and child s

times for a week and in other places for two weeks, to work primarily with the parent We try to teach the mother what she can do with the child This is in addition to what many of these parents have already learned from the John Tracy Clinic correspondence course Even though they have taken that course they like some additional help in interpreting the whole thing Whereas we may be able to give them one or two weeks a year, it would be much better if it could be done more frequently

# VII IMPROVEMENTS IN ELECTROACOUSTIC INSTRUMENTATION

In many of the earlier presentations that described particular tests the participants mentioned the equipment employed but in only one case was there discussion of possible improvements in equipment to make the test more generally available This exception was in the case of the average response computer and associated equipment to record cortical evoked responses Dr Scott discussed specifically the development of a simplified system

No mention was made of possible improvements in group hearing aids or other systems for providing amplified sound for children other than individual electrically-operated hearing aids

### *Improvements in Hearing Aids*

The shortcomings of and possible improvements in wearable hearing aids for young children was the final item on the agenda of the conference The discussion moved rapidly and seldom exhausted any single topic The Editor has here condensed the discussion because its chief value will be to provide an agenda for some future conference or research project Actually as Dr Barr pointed out it would have been impossible to do much more than prepare this list of topics because no engineers were present Any meaningful discussion of improvements must include an electroacoustic engineer who can give some idea as to whether a particular suggestion is feasible and if it is feasible then at what expense or with what necessary compromises

CHAIRMAN The last item on our agenda is possible improvements in hearing aids particularly hearing aids for young children The tentative list is as follows

| | |
|---|---|
| Smaller size | Less expensive |
| More rugged | More acoustic output |
| Waterproof | Better earmolds |
| Better electroacoustic characteristics | |

Dr Ling in his presentation has already put in a word for extending the frequency characteristic to include low frequencies This extended characteristic may not be required in all instruments but it is definitely

times for a week and in other places for two weeks, to work primarily with the parent. We try to teach the mother what she can do with the child. This is in addition to what many of these parents have already learned from the John Tracy Clinic correspondence course. Even though they have taken that course they like some additional help in interpreting the whole thing. Whereas we may be able to give them one or two weeks a year, it would be much better if it could be done more frequently.

W HARDY I have one idea about the prevalence in the United States I have heard Professor Ewing's figure of 1 2 per thousand However, if we add up all the children with hearing losses sufficient to interfere with their daily classroom learning, we should multiply that figure by 10 Then there are others who need these instruments who are not always counted, such as the cerebral palsy children and others with multiple handicaps

DAVIS I still wonder, even if we multiply Sir Alexander Ewing's figure by 10, whether it is large enough to provide the incentive for the hearing-aid industry to do this job of re evaluation and redesign This is a major project, and I believe that our Federal Government is the only likely source for financing it Remember that the Harvard report of 1948 was based on a wartime project, directed toward the rehabilitation of veterans

CHAIRMAN Discussion could continue but the time has come when we must adjourn Let me thank the participants for a very interesting and valuable set of contributions and discussions

A EWING I am sure that all of the participants desire to thank the organizers and sponsor of this conference very, very sincerely for a wonder fully successful job of the highest importance most efficiently carried out

## *Closing Remarks by Mr. Fox*

At the adjournment of the session Mr Fox thanked both the organizers and the participants of the conference and expressed his deep satisfaction with the progress that had been made He mentioned the acceleration of progress that had been achieved by the direct exchange of information He said in part, "I am sometimes impatient with delays but I do recognize the importance of getting all the facts before you come to a conclusion I am sure that as the result of this meeting there will be a speeding up of the development of methods and procedures Of one point *I feel sure* the sooner we detect impaired hearing the better, and the sooner we get at the training of the defective child the better And if there is need for another such conference as this in one or two years I shall be glad to do my part to make it possible'

★

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S U P P L E M E N T U M 203

# EFFECT OF SOME OTOTOXIC DRUGS UPON THE PATTERN AND INNERVATION OF COCHLEAR SENSORY CELLS IN THE GUINEA PIG

BY
AARNO KOHONEN

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

# EFFECT OF SOME OTOTOXIC DRUGS UPON THE PATTERN AND INNERVATION OF COCHLEAR SENSORY CELLS IN THE GUINEA PIG

FROM THE EAR NOSE AND THROAT DEPARTMENT UNIVERSITY OF GÖTEBORG SWEDEN (HEAD PROF GÖSTA HERBERTS MD) FROM THE HELSINKI UNIVERSITY OTOLARYNGOLOGICAL HOSPITAL, HELSINKI FINLAND (HEAD PROF URPO SIIRALA MD) AND FROM THE DIVISION OF NEUROLOGICAL SCIENCES US NAVAL SCHOOL OF AVIATION MEDICINE PENSACOLA FLORIDA USA (HEAD HARLOW W ADES PHD)

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 208

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*by*

AARNO KOHONEN

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# I INTRODUCTION

In recent years the rapid development of new antibiotics has produced several drugs which, while otherwise clinically valuable, have the unfortunate side effect of causing serious damage to the auditory mechanism, thus limiting their clinical use An extensive and growing literature has accumulated over the past decade consisting of reports of studies on relative ototoxicity, critical dosage, and auditory damage produced by this group of drugs These studies include many based on audiological follow up of drug administration and also a number of animal experiments in which the histologic effects on the inner ear have been assessed in relation to drug dosage and functional deficit

The experimental approach has yielded a considerable mass of data published in several reports While there is a certain amount of disagreement, the consensus of these reports points to the sensory cells of the organ of Corti as the site of primary damage, however, precise information on the exact location of damaged hair cells as regards the respective coil and row of hair cells is generally lacking in these reports Likewise, surprisingly little information is available relating to the effect of the ototoxic antibiotics on neural elements of the organ of Corti Several reports describe drug induced damage to spiral ganglion cells and a few recent papers have dealt with drug-induced degeneration of nerve endings on hair cells as revealed by electron microscopy, however, none of these gives any information on the general pattern or distribution of nerve fiber and nerve ending degeneration in the organ of Corti These informational gaps are undoubtedly due to limitations of conventional histologic methods

A new approach to this sort of study is offered by the surface preparation technique developed by *Engstrom* and his associates used in combination with phase contrast and electron microscopy and with a new nerve staining method The surface preparation technique is described in preliminary reports by *Engstrom* et al (1964) and by *Hawkins* and *Engstrom* (1964) The studies reported in the present paper were prompted by the belief that the evaluation of cochlear damage by ototoxic drugs could be greatly advanced by application of this combination of methods to the structural analysis of the damaged cochlea The paper will deal with parallel analyses of the general pattern of cellular damage and that of the neural elements, thus making it possible to interrelate the degeneration of sensory cells, nerve fibers, and nerve endings in groups of guinea pigs subjected to various ototoxic drugs in systematically varied dosages In addition, there will be considerable emphasis devoted to the description of a reproducible graphic method of representation of such data

# II REVIEW OF LITERATURE CONCERNING OTOTOXIC ANTIBIOTICS

## A. Neomycin

*Neomycin was isolated in 1949 by Waksman* and *Lechevalier* as a result of their search for new antibiotics lacking the resistance inducing effect of streptomycin in bacteria (*Waksman*, 1953) Neomycin is a mixture of three closely related basic antibiotics It *is* mainly bactericidal, has an unusually broad antibacterial spectrum effective against several Gram-negative bacteria, including Pseudomonas, almost all staphylococci, and has a tuberculostatic effect

The earliest clinical reports (*Carr* et al 1950, 1951, *Waisbren and Spink*, 1950) showed that neomycin had a considerable ototoxic effect *Waisbren* and *Spink* reported on 65 patients who had received neomycin treatment over a relatively short time Hearing loss was found in four patients, all of whom also had some kind of renal disease *Carr* et al reported results on six tuberculous patients who had received long term neomycin therapy Four showed hearing loss and the authors concluded that neomycin was too ototoxic to permit its use for long term treatment

Severe hearing loss resulting from neomycin therapy was subsequently observed by several investigators (*Goldner*, 1958, *Kortge-Stoppler* and *Mittag*, 1958, *Greenwood*, 1959, *Leach*, 1962) To summarize the observations reported in these papers, the hearing loss often occurred days or weeks after administration of the drug had been stopped It began characteristically at the higher frequencies and, in many cases progressed to total deafness Hearing loss was definitely more pronounced than vestibular disturbance Only minor vestibular disorders were found associated with total deafness Patients with renal disorders, even of slight degree, were especially sensitive to the ototoxic effect of neomycin, total deafness often occurring in such patients after treatment, even with small dosage

Because of its broad antibacterial spectrum and low absorption rate from the intestine neomycin has been recommended for use as an intestinal antiseptic to be given by mouth or rectum, however, even by these routes of administration, large doses have occasionally resulted in cochlear damage (*Halpern* and *Heller*, 1961, *Fields*, 1964) Long time aerosol therapy in patients suffering from chronic bronchitis has also been known to cause hearing loss (*Fuller*, 1960, *Helm*, 1960)

Animal experiments by *Hawkins* (1952) showed by electrophysiological measurement that neomycin administered parenterally damages the cochlea in cats Further studies in rats (*Courvoisier* and *Leau*, 1956) and guinea pigs (*Osada*, 1962, *Tyberghein*, 1962) confirmed *Hawkins'* conclusion that neomycin has a very pronounced [illegible] effect on experimental animals In this respect, it is one of the strongest of [illegible] antibiotics belonging to the Streptomyces group The first report of cochlear damage induced by neomycin and studied histologically was published by *Ruedi* and his collaborators in 1955 They treated guinea pigs with neomycin and the animals

were sacrificed six weeks after the disappearance of the pinna reflex They found that in the three basal coils of the cochlea all of the outer and inner hair cells had disappeared some isolated hair cells remaining in the apical coil Destruction was greatest at the beginning of the second coil where the organ of Corti had been replaced by a flat layer of epithelial cells resting on the basilar membrane In addition the supporting elements of the organ of Corti had undergone varying degrees of degeneration in the basal part of the cochlea These changes extended to the stria vascularis Damage of neural elements was also reported as expressed in marked reduction of the number of nerve fibers in the spiral osseous lamina and number of ganglion cells in the spiral ganglion in regions corresponding to the location of the most severe degeneration of the organ of Corti

*Hawkins* and *Lurie* (1955) compared the ototoxic properties of dihydrostreptomycin and neomycin in cats applying both electro-physiologic and histologic methods. In one animal treated with neomycin histologic studies showed degeneration of outer hair cells in the basal coil and in the lower part of the second coil Scattered loss of outer hair cells was present also in the upper part of the second coil and in the third coil Inner hair cells were considered normal A second cat treated with a larger dose of neomycin showed complete degeneration of cochlear sensory cells but no collapse of the organ of Corti was found as in the animals of *Ruedi* and his collaborators (1953) Neither was there any damage to spiral ganglion cells or to nerve fibers in the osseous lamina In this second animal slight damage was noted in the vestibular sensory epithelia

*Riskaer* and his co workers (1956) studied the damage of inner ear sensory cells caused by neomycin and compared it with the damage caused by streptomycin vio mycin and polymyxin B A daily dose of 50 mg neomycin per kg body weight in 50 days or less did not result in any histologically discernible changes Doses of 100 mg/kg body weight in 30 days and 150 mg/kg body weight in 60 days resulted in extensive damage of the cochlea In the middle coils the organ of Corti was completely destroyed while in the basal and apical coils its contour was discernible with partly or completely degenerated atrophic hair cells The spiral ganglion showed extensive atrophy the vascular stria was narrowed No changes were found in vestibular epithelia although functional disorders of slight degree could be detected in some animals Histologic study of the brains of the animals was also carried out Chromatolytic and chromophobic changes in the nuclei and atrophy of the ganglion cells with proliferation of the surrounding glia was found in the acoustic tubercles The authors also reported slight degeneration of the ganglion cells in the Deiters nucleus

*Olivieri* and *Rossi* (1958) compared the ototoxic properties of neomycin sulphate and neomycin glucuronate in guinea pigs In all of these animals the most pronounced histologic damage was found in the two lower coils of the cochlea the outer hair cells showing more damage than the inner Large daily doses of the drugs caused severe destruction of the organ of Corti and some degeneration of the spiral ganglion cells whereas administration of small doses for a long time resulted in considerable degeneration of the spiral ganglion with less marked damage in the

organ of Corti The glucuronate salt of the drug was found less ototoxic than its sulphate

*Lindsay* and his co workers (1960) studied the temporal bones of a patient who died of bacterial endocarditis He had been given a total dose of 18 g of neomycin after which he developed a severe hearing loss In both cochleas inner hair cells were found to be completely absent The outer hair cells were somewhat better preserved their degeneration being estimated at 60—100 % depending on location The most severe damage was found in some parts of the basal and apical coils, where some loss of Deiters' cells and pillar was seen in addition to loss of hair cells No clear cut changes were found in the stria vascularis or in the vestibular epithelia

*Friedmann* and *Bird* (1961) studied the effects of neomycin and kanamycin on isolated otocysts of fowl embryos The cells of sensory areas were affected primarily, but some disintegration of nerve axons was also observed The type of damage was similar for both drugs, neomycin being definitely the more toxic of the two

## B Kanamycin

Kanamycin was isolated in 1957 by *Umezawa* from a culture of a Streptomyces strain called Streptomyces kanamyceticus by the discoverer (*Umezava*, 1958) The chemical structure of the new antibiotic was determined by *Cron* and his associates (1958) It is chemically closely related to neomycin and less closely to streptomycin It is effective against a variety of different Gram positive and Gram negative bacteria Proteus and Pseudomonas among them, against some strains of staphylococci resistant to other antibiotics, and has a distinct tuberculostatic activity

It was to be expected that kanamycin as a member of the group of Streptomyces antibiotics, would have certain ototoxic and nephrotoxic properties This soon became evident as numerous clinical publications reported on side effects of kanamycin therapy (*Frost* et al 1958/1959, *Lustberg* and *Hamburger*, 1959, *Lecca* et al 1959, *Naunton* and *Ward*, 1959, *Partsch*, 1961, *Alfthan* et al, 1962, *Haapanen*, 1963) Careful audiologic control of the patients proved to be necessary during treatment with kanamycin especially if long term therapy was used e g, in tuberculosis The hearing loss from kanamycin therapy first appears in the high frequency range A case of partial recovery from kanamycin induced hearing loss, when administration of the drug was immediately stopped at the first signs of cochlear damage, has been reported (*Hawkins*, 1959) 'Delayed' ototoxicity, i e, hearing loss appearing several weeks after the administration of the drug as found in certain patients treated with neomycin and dihydrostreptomycin, has not occurred after kanamycin Previous renal disorders greatly increased the risk of cochlear damage, so that administration of a few grams was sometimes followed by total deafness Several authors have also reported vestibular disorders from kanamycin *Hawkins* (1959) could not prove the presence of recruitment as a characteristic sign in cases with hearing impairment caused by kanamycin in man but other investigators (*Sataloff* and *Menduke*, 1964) have found recruitment in hearing loss resulting from administration of kanamycin

Using electrophysiologic methods and cats as test animals, *Hawkins* (1959) proved that in some animals kanamycin treatment selectively depressed cochlear microphonics, while action potentials were still preserved In other animals both cochlear micro phonics and action potentials were depressed Later studies using similar techniques (*Tyberghein*, 1962, *Ouada*, 1962) confirmed the impression that kanamycin has a pronounced toxic effect on the cochlea of experimental animals

In the paper quoted above *Hawkins* also published histologic pictures from animals treated with kanamycin In one of the cats receiving 'kanamycin A", outer hair cells were absent in the lower coils of the cochlea but intact in the apex The inner hair cell nuclei were absent throughout the cochlea and pycnotic changes of spiral ganglion cells were also present Treatment with 'kanamycin B' resulted in more pronounced damage in the cochlea In these cats the organ of Corti had completely disappeared in the basal coil and in the second coil it was represented only by a flattened layer of cells on the basilar membrane Corresponding to the localization of the most extensive hair cell damage, ganglion cells and nerve fibers in the spiral osseous lamina were completely missing in the basal coils of the cochlea and only fragments of degene rated nerve fibers were left in the modiolus In guinea pigs, a similar localization of cellular loss was found without neural damage Rats seemed to be less susceptible to kanamycin, but proportionately larger doses of the drug resulted in cellular and neural damage similar to that seen in cats

*Ward* and *Fernandez* (1961) found that, if guinea pigs were treated with toxic doses of kanamycin, an almost selective destruction of outer hair cells could be observed The severity of the destruction was most pronounced in the basal coil de creasing towards the apical turns In the basal coil scattered loss of inner hair cells was also seen, as well as a considerable loss of Deiters' cells In one specimen a definite decrease in numbers of ganglion cells and fibers of the auditory nerve was evident Histologic examination of the animals' central nervous system revealed patchy, disse minated, rarified areas in the cerebellum and in the brain stem

*Mesolella* and *Costa* (1960) also reported cochlear damage in guinea pigs after kanamycin Their results were in agreement with those of *Ward* and *Fernandez* with respect to the organ of Corti, but they found only slight changes in the spiral and vestibular ganglia and in the central nervous system

*Catalano* et al (1961) showed in kanamycin treated guinea pigs, extensive hair cell damage and loss of ganglion cells in the two basal coils of the cochlea *Darrouzet* and *De Lima Sobrinho* (1962) found, also in guinea pigs, a selective damage of outer hair cells in the basal turn with slight changes in the supporting cells Later *Darrouzet* (1963) studied the protective properties of vitamin B complex and amino acids against kanamycin He reported that pre-medicated animals showed definitely less damage than the untreated controls

In 1962 *Beck* and *Krahl* published a report on cochlear changes in kanamycin-treated guinea pigs They made use of the microdissection technique of *Neubert* (1950) and studied the structural changes in the hair cell nuclei, reporting disarrangement of the nuclei of the first row of the outer hair cells At a later stage a similar disarrangement was also found among the nuclei of the second row of the outer hair

cells, the third row remaining undisturbed These early nuclear changes were followed by swelling und ultimate disintegration of the nuclei, so that in some specimens the nuclei of the first and second row had completely disappeared, the nuclei of the cells of the third row still being visible At a later stage similar changes were found in the inner hair cells, though it was not possible to establish definitely the temporal relations of inner to outer hair cell damage Degeneration of Deiters' cells apparently began at the same time as that of the inner hair cells The destruction was most marked in the basal part of the cochlea, where profound structural changes in the stria vascularis and in the spiral ganglion were also found *Histologic examination of the animals'* brains showed almost selective degeneration of the dorsal cochlear nucleus

*Reddy* and *Igarashi* (1962) published a series of histologic pictures from cats treated with kanamycin They suggested that the supporting cells, especially the cells of Claudius, would be most sensitive to kanamycin and that the sensory cells would degenerate later At a late stage of degeneration they found a complete loss of the organ of Corti and of spiral ganglion cells in the basal coil

In guinea pigs *Ardouin* et al (1963) found loss of outer hair cells in the basal coils and pycnotic changes in outer hair cell nuclei in the second and third coils No changes could be discerned in the inner hair cells, supporting cells, or neural elements

Combined light and electron microscopic studies of cochlear changes in kanamycin-treated cats were reported by *Farkashidy* et al in 1965 Their light microscopic findings agreed with those of earlier authors in that the most extensive damage was seen in the basal coil, the outer hair cells showing more effect than the inner Under the electron microscope, the inner hair cells appeared intact, while the outer showed marked changes These included absence of sensory hairs and cuticular plate, and supranuclear clumping of mitochondria and endoplasmatic reticulum Likewise, the nerve endings under the inner hair cells were normal in appearance, whereas those of the outer hair cells were swollen and showed mitochondrial clumping The nerve fibers of both inner and outer spiral bundles were well preserved The authors suggested that the supranuclear localization of the early degenerative signs might be the result of high concentration of the drug in the endolymph

*Two human cases of histologically verified, kanamycin induced changes in the* inner ear have been reported (*Benitez* et al, 1962, *Jorgensen* and *Schmidt*, 1962) As in the case of animal experiments the greatest damage was seen in the outer hair cells of the basal coil *Jorgensen* and *Schmidt* also noted some loss of ganglion cells in the lower part of the spiral ganglion

Using *Engstrom*'s method of surface preparation of the organ of Corti, *Hawkins* and *Engstrom* (1964) studied the cochleas of kanamycin-treated guinea pigs The earliest change seen by this method is a disarrangement of the W-pattern of the sensory hairs on outer hair cells This is then followed by further degenerative changes leading to complete loss of the cell The outer hair cells of the basal coil and the first row of outer hair cells of the third coil showed greatest sensitivity to the drug Only scattered loss of inner hair cells and pillar cells was seen Increased dosage brought about further degeneration of basal coil supporting cells, especially the cells of Claudius situated peripheral to the third or outermost row of outer hair cells

# C Framycetin

Framycetin was isolated by *Decaris* in 1953 It is produced by a strain of Streptomyces lavendulae Its chemical structure is not fully known, but appears to be related to that of streptomycin Its action is that of a broad spectrum antibiotic effective against a wide variety of Gram positive and negative bacteria No naturally occurring strains of staphylococci resistant to the drug have been encountered yet Because of its toxic effect on inner ear and kidney framycetin is not recommended by the manufacturer for parenteral use although it has been found useful for local application It can also be used in certain intestinal infections since it is poorly absorbed from the intestine The antibacterial and pharmacologic properties of framycetin have been discussed by several authors (*Decaris* 1955 *Fairbrother* and *Williams* 1958, *Maccabe*, 1959) none of whom give any data on the drugs ototoxicity *Massenat—Deroche* (1954) described severe toxic side effects on the ears and kidneys of a group of patients who had received a daily dosage of 0 750—1 0 g of framycetin, and suggested that further clinical experiments with parenteral administration should be avoided and the drug reserved for topical therapy and for very severe infections against which other antibiotics had proved ineffective

No animal experiments with framycetin have been reported For this reason it was deemed of interest to include it in the experiments reported in this paper and to examine its effects on the cochlea despite the fact that the drug is not used clinically for parenteral administration

# D Summary of previous findings on histologic changes in the cochlea caused by the antibiotics studied

There is general agreement that the main ototoxic effect of neomycin centers upon the organ of Corti This applies also to the two studies dealing with concomitant changes in the spiral ganglion and the central nervous system (*Ruskaer* et al 1956 *Olivieri* and *Rossi*, 1958) Animal experiments have shown that the inner hair cells are more resistant than the outer to the ototoxic effects of the drug although one report seems to indicate that the reverse is true in man (*Lindsay* et al 1960) All authors without exception agree that the basal portion of the cochlea is the most vulnerable however with increasing dosage the degeneration may be extended to include considerable portions of the organ of Corti Most authors agree that the sensory cells are most sensitive to the drug however several also noted extensive degeneration among supporting structures nerve fibers of the osseous spiral lamina and the spiral ganglion cells although such changes seem to require a longer time to develop than do changes in the sensory cells

All of the data on kanamycin induced cochlear damage point to the basal coil as the primary site of predilection and to the outer hair cells as markedly more vulnerable than the inner As is the case with neomycin destruction of supporting cells and loss of spiral ganglion cells in the basal part of the cochlea have been described by

several authors as late or advanced phenomena of kanamycin intoxication, which is in agreement with observations on human temporal bones A few studies also report kanamycin-induced changes in the dorsal cochlear nucleus With respect to the pattern of hair cell damage, both *Beck* and *Krahl* (1962) and *Hawkins* and *Engstrom* (1964) agree that the hair cells of the first outer row are the most sensitive to the drug *Hawkins* and *Engstrom* have contributed some details on the pattern of degeneration of individual sensory cells The single report on kanamycin damage to intrinsic neural elements of the organ of Corti is that of *Farkashidy* et al (1963), who described degenerative changes in nerve endings on outer hair cells as seen under the electron microscope

No experimental histologic information on the ototoxic effect of framycetin has been reported The evidence on this drug has so far been confined to reports of clinical data indicating an adverse effect on hearing

# III PLAN OF PRESENT INVESTIGATION

Despite the many studies on ototoxicity of antibiotics as reviewed in the preceding sections it was the author's belief that much additional information was needed to clarify the basic process underlying cochlear destruction and progressive hearing loss following treatment with these drugs The availability of new methods for study of the cochlea and for comprehensively registering the damage, made possible a fresh attack on several aspects of the problem which have not been studied previously, and on details of the degenerative process at progressive stages The following questions have been given special attention and serve to outline the principal direction of attack

1 What happens to the individual sensory cells of the organ of Corti and do they undergo characteristic sequential stages in their degeneration?

2 Is there a systematic pattern of degeneration within the cochlea as a whole, and if so, does this correspond with any pattern of structural differences within the normal cochlea?

3 If there is a systematic pattern of degeneration, how can its details best be recorded and demonstrated?

4 Do intrinsic neural elements of the cochlea show a characteristic pattern of degeneration and if so does it parallel that of the sensory cells?

# IV MATERIAL AND METHODS

## A. Experimental animals

The histologic material consisted of the cochleas of 58 guinea pigs used in the drug experiments plus a large number of similarly prepared guinea pig ears already on hand in the laboratory The experimental group of 58 animals include only those given ototoxic drugs They were obtained from a single source, most were non albino and weighed 250—350 g at the start of the experiments Only animals showing marked pinna reflex were accepted

Control material was furnished by the normal ears available in the laboratory which formed the material for a structural analysis of the normal cochlea, in which the author collaborated The results of that study are to be published by *Engstrom*, *Ades*, and *Andersson* in a forthcoming monograph That study and monograph form the technical background for this research

## B. Drugs

The neomycin preparation used was Mycifradin Sulphate manufactured by Upjohn Company The drug was supplied in vials containing 0 5 g of dry substance corresponding to 0 35 g neomycin base According to the manufacturer the preparation consists of 90—95 % neomycin B, 5—10 % neomycin C and contains no neomycin A The contents of a vial were dissolved in 2 5 ml physiologic saline to make a solution *of 200 mg neomycin per ml*

The kanamycin used in the experiments was manufactured by Meiji Seika Kaisha Ltd Tokyo, and supplied in vials of 1 g dry kanamycin sulphate It was dissolved in 4 2 ml distilled water to make a solution containing 200 mg kanamycin sulphate per ml of solution

The framycetin preparation used was called Soframycin, manufactured by Roussel Laboratories The dry weight contents of a vial, 1 g framycetin sulphate, was dissolved in 5 ml physiologic saline to make a solution of 100 mg framycetin sulphate per ml

## C. Plan of drug administration

[illegible] uinea [illegible] e treated in groups of 4 to 10 animals The antibiotics were inj[illegible] ubcutan[illegible] daily until the planned total dose had been given The site of [illegible] ction w[illegible] ly in order to avoid local damage to the subcutaneous

tissue The animals were weighed daily during the treatment and the daily dose of drugs calculated in relation to the actual weight The threshold of the pinna reflex was tested daily with a high frequency Galton whistle as a rough determination of the progress of hearing loss The daily weighing and testing of hearing were continued until about a week after the last injection and in the case of animals which were allowed to survive longer repeated on the day they were sacrificed The gain or loss of weight served as an indicator of the animals general health as did their behavior liveliness and appetite

Several animals died during or shortly after the treatment All of these were discarded If signs of middle ear infection were observed upon opening the tympanic bulla those cochleas were also discarded

## D Histologic methods

### 1 FIXATION AND SURFACE PREPARATION OF THE COCHLEA

The guinea pigs were killed by decapitation The lower jaw was cut away with scissors the bony tympanic bullae exposed from below and widely opened The temporal bones were dissected free from the skull the stapes removed the round window opened and a piece of bone at the apex of the cochlea was broken away The fixation fluid 1 5 % veronal buffered osmium tetroxide solution at a temperature of 2—4°C was dropped into the opened apex of the cochlea by means of a micropipette so that it could be seen running through the cochlea and out through the windows Use of this technique minimized post mortem changes as no more than 1 to 2 minutes intervened between death of the animal and application of the first drops of the fixation fluid

The specimens were left for one to two hours in the fixation fluid under refrigeration following which they were washed for one hour in physiologic saline solution and were then ready for further dissection If not dissected on the same day the specimens were left overnight in the refrigerator immersed in 70 % alcohol The final dissection was done under a binocular dissecting microscope with magnifications of 6 × to 10 × the specimen being immersed in physiologic saline solution or 70 % alcohol Using dental instruments the bony capsule of the cochlea was broken away over a considerable area and the spiral ligament together with the stria vascularis removed thus uncovering the coils of the cochlea and the organ of Corti With small watchmaker s tweezers Reissner s and tectorial membranes were removed and the desired sample from each turn of the organ of Corti was dissected free (Fig 1) The samples so obtained were then placed on glass slides and covered with a drop of glycerin An ordinary cover glass was then carefully placed over the specimen which was now ready for examination under the phase contrast microscope




FIG. 1. — *General view of right cochlea of guinea pig. The bony capsule has been removed. In the upper coils of the cochlea osmiophilic particles can be seen in the Hensen cells. They are of great value for orientation in the specimen. At the basal end of the cochlea the organ of Corti (OC) can be seen in its normal position. Many details of innervation and structure may be seen even at this low magnification. Arrows in each coil of the organ of Corti indicate portions which we routinely dissect free for study as surface specimens. 35 ×*

Many of the cochleas were examined as sections under phase contrast or electron microscopy For this purpose they were fixed in the manner described above and after washing were dehydrated through a series of alcohols of increasing concentration The whole cochleas or parts of them were then embedded in epoxy resin or acrylate Sections for phase contrast microscopy for ordinary survey studies were cut by hand with a razor blade or on a standard microtome For electron microscopy sections were cut on an LKB Ultrotome The latter instrument was extensively used also in preparing sections for closer phase contrast study

## 2 IMPREGNATION OF NEURAL ELEMENTS OF THE COCHLEA

For the study of the innervation of the organ of Corti a modification of the osmium tetroxide zinc iodide technique published by *Maillet* (1963) has been developed and used

The fixation — staining solution consists of two parts

A 1 5 % veronal buffered osmium tetroxide solution

| | |
|---|---|
| B Twice sublimated iodine | 5 g |
| Zinc powder | 15 g |
| Distilled water to make | 200 g |

In the presence of an excess of metallic zinc the latter solution contains zinc iodide The excess of metallic zinc is removed by filtration immediately before use Three parts of fluid A are then mixed with 8 parts of fluid B upon which the solution suddenly turns brown and within an hour a precipitate begins to form in the solution One to four guinea pig cochleas could be conveniently stained in 15 20 ml of the solution

After decapitation the temporal bones were dissected free and the tympanic bulla of each was opened The specimen was then transferred to a vessel containing freshly mixed solution of the fixative — stain There the stapes was removed the round window opened the bony shell covering the tympanic side of the basal coil removed and a narrow zone of the bony cochlear capsule removed beginning at the top and continuing downwards to the basal coil All this was done with the specimen lying submerged in order to avoid drying of the cochlea The specimen was carefully turned several times in the jar to permit the stain to reach all parts of the cochlea Wide opening of the cochlea and turning of the specimen in the solution have been found to be essential for good results

The specimens were impregnated in the solution described above for 17—20 hours washed in physiologic saline solution for one hour and dehydrated through increasing concentrations of alcohol The surface specimen of the organ of Corti is prepared in absolute alcohol in the way described earlier The samples of the organ of Corti are passed through xylol and mounted on glass slides in a suitable medium such as Canada balsam for examination by light microscopy

Part of the nerve impregnated cochleas underwent the same process of epoxy resin or methacrylate embedding and sectioning as those fixed with osmium tetroxide alone They were examined under light or electron microscope Some specimens were also decalcified to permit easier sectioning and this has yielded excellent results in cases where it was desirable to have sections of the intact cochlea

# E. Normal structure of the organ of Corti in surface specimens

## 1 CYTOARCHITECTURE OF THE ORGAN OF CORTI

The normal structure of the organ of Corti as revealed by conventional radial sections is well known to everyone engaged in this field of histologic work. The examination of surface specimens offers a different view of this organ. Unstained and unsectioned parts dissected out from the coils of the cochlea, previously fixed in osmium tetroxide, are studied by phase contrast microscopy. Despite the thickness of the acoustic papilla (about 100 μ), it is possible to focus on different levels and thus to make "optic sections" through the specimen. At a given level, details of the organ of Corti can be seen with amazing clarity. By turning the micrometer screw of the microscope, level after level can be brought into focus, studied, and photographed. It is thus possible to start at the upper surface of the specimen and study individual hairs and the general arrangement of the hairs, then the field of vision can be focused on the cuticular plates, the infracuticular region, the nuclei of the sensory cells, and finally on the basilar membrane and the tympanic cover layer. Thus, many details in a chosen portion of the organ of Corti can be scrutinized. One of the outstanding virtues of the method is that the cellular components retain their exact interrelation, so that it is possible to discern small irregularities, malformations, cellular losses or systematic degeneration by direct observation without need of reconstruction.

A good idea of the method is achieved by studying Figures 2–4 which represent different levels from unsectioned specimens of the organ of Corti. In Figure 2 the plane of the "optic section" is at the level of the hairs of a series of outer hair cells, and the W-arrangement of the hairs is clearly seen. Above the first row of the outer hair cells the heads of the outer and inner pillar cells are seen forming a transverse ribbon. In Figure 3 the level of focus is somewhat deeper, passing through the cuticular plates of the outer hair cells of the three rows. This view shows clearly how the outer hair cells form a continuous, strictly regular mosaic, where each cell has its determined place in the pattern in relation to its neighboring sensory and supporting cells. The numbers 1, 2 and 3 indicate the three rows of outer hair cells. In the upper part of the picture the level of focus is in the plane of the sensory hairs of the inner hair cells. Individual hairs can be discerned on many of the cells, and the hairs are found to be arranged in two to three parallel, slightly curved rows on each cell. In the lower part of the picture nuclei of the Hensen cells are seen peripheral to the outer hair cells. In the upper part of the cochlea the Hensen cells contain osmiophilic particles visible as black dots. In Figure 4 the level of focus is in the plane of the cuticular surface of the inner hair cells. They form a single row above the ribbon of the heads of outer and inner pillars. Each inner hair cell is separated from the neighboring hair cells by the narrow phalangeal processes of inner phalangeal cells. The level sections the outer hair cells of the first and second rows slightly below the cuticular plates so that the basal bodies become visible, located at the angle of the "W" of the hairs in a cuticle free region.



FIG 2 — *Surface preparation from tl e upper part of the basal coil of gu nea p g cochlea Tl e sensory hairs form a W figure on each outer hair cell The angle betu een the branches of tl e W is r iore tl an 90° It the basal extren ity this angle is about 120° decreas ng to 60°— 0° at the apex Irrou s indicate t io superm ı nerary outer hair cells forn ing a partial fourth row Phase contrast 1345 ×*

The general arrangement of the cellular structures is similar in all parts of the cochlea although characteristic differences occur from coil to coil Thus the outer hair cells of the basal coil have smaller volume and their cuticular plates a more kidney shaped form than those of the outer hair cells of the middle and apical coils The angle between the arms of the W formed by the hairs on the outer hair cells diminishes from the basal coil towards the apex of the cochlea On the basis of such differences the examiner can tell at a glance whether a given specimen is taken from the basal middle or apical part of the cochlea The details of the normal cellular pattern are thoroughly discussed by *Engstrom Ades* and *Andersson*

As already mentioned any desired level of the specimen can be examined with great ac ı ra y and the structure e g of sensory or supporting cell nuclei studied howev r tl e surface view of the organ of Corti as shown in the figures is of more imı rtance when evaluating the gross pattern of cellular damage caused by noxious agents In contrast to conventional serial sections it provides the examiner with an lea of the whole architecture of the organ and in the case of systematic pathologic changes with a panoramic view of the pattern of damage




FIG 5 *Si rface preparat on fro n second co l of gu nea p g cocl lea 1 2 and 3 nd cate tl e f rst second and tl ird ro s of o ter l a r cells H nd cates l a rs on a long ro v of i iner l a r ell Tl ese cells a e not in foc s except for tl e ha rs O i left it can l e seen tl at tl e l a rs l a t two ro s P l eads of inner and outer p lla s HeC Hensen cells Pl alangeal e a ly een l et een tl e oute l a r cells Pl ase contrast 1590 ×*

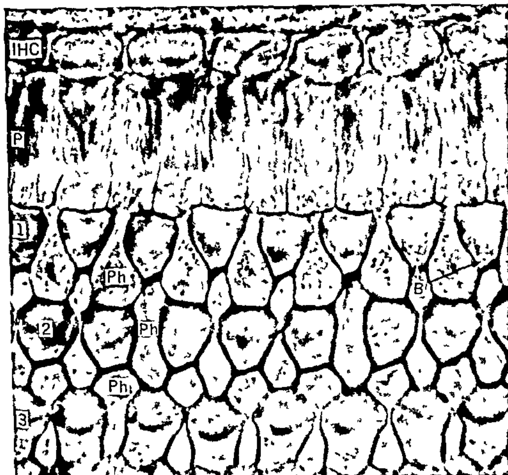



FIG 4 — *Surface preparation from middle coil of guinea pig cochlea IHC = inner hair cells P = heads of inner and outer pillars Ph — phalanges of Deiters cells between outer hair cells Note the basal body region (B) in the first row of outer hair cells Phase contrast 1560 ×*

## 2 THE COCHLEOGRAM

By aid of the surface specimen technique it is possible to survey large portions of the organ of Corti in normal as well as in pathologically altered cochleas It is easy to localize individual cells or groups of cells undergoing degeneration and because of the orderly arrangement such cells can easily be registered in relation to adjacent normal cells In this way cellular damage or cellular loss can be exactly recorded in individual experimental animals If the specimens are taken from corresponding region[illegible] of the cochleas a direct comparison of the extent of damage can be made from one animal to another As will be discussed below the sensory cells degenerate after exposure to ototoxic antibiotics and the degeneration follows a characteristic course ending with what we call collapsed cells These degenerating

cells stand out very distinctly in contrast to cells of normal appearance under the phase contrast microscope and can be plotted quickly and accurately in a simple diagram, which, following *Engstrom*'s description, we call a "cochleogram". The cochleogram, thus, is a schematic graphic presentation of the arrangement of the sensory cells, where each cell, normal or damaged, *has its correct place in the pattern*. The "normal" cells are registered as an open circle (O) and the "degenerated" cells as a solid circle (●). The three lower rows represent the outer hair cells and the upper single row the inner hair cells (Fig. 15).

In cochleograms from normal guinea pigs, certain deviations from the generally strict, geometric pattern can be found. In general, there are three rows of outer and one row of inner hair cells. A few supernumerary outer hair cells are often found in the third row and now and then four rows of cells can be observed for short distances. Extra cells are found less often in the second and first rows. Supernumerary inner hair cells are very uncommon in the guinea pig, but may be found in some other species (*Retzius*, 1884, *Engstrom, Ades*, and *Andersson*).

In addition to supernumerary cells, irregularities of pattern of another type can be observed in which occasional cells are missing, i.e. have failed to develop. Such gaps in the pattern are most frequent towards the upper end of the cochlea in the third and second rows, in that order. Both supernumerary and "missing" cells can easily be plotted in the cochleogram. As there are sometimes difficulties in distinguishing between "missing" cells and damaged cells, we have elected arbitrarily to register both as damaged in our cochleograms from animals exposed to antibiotics. The error introduced hereby seems to be of negligible importance.

As the cochleogram is a diagrammatic representation of the sensory cell damage, the cells registered in it must be sharply divided into two groups, the "normal" and the "degenerated" cells. The cells showing the characteristic figure of a "collapsed" cell, discussed in the next chapter, were plotted as "degenerated" cells as well as the missing cells mentioned above. Other cells were plotted as "normal". This means that the "normal" cells in the cochleograms of this study include cells showing slight pathologic changes that will be discussed in the next chapter, e.g. swelling of the nuclei or disarrangement of the pattern of the sensory hairs. Such an arbitrary division of the cells into two groups is, of course, in a way, incorrect, as the degeneration follows a gradual course, however, it will considerably facilitate the presentation of the material. On the other hand, a normal appearance by the microscopic method used does not necessarily imply that the cell functioned normally just before the animal was sacrificed.

In practice the recording of the sensory cell damage in the cochleograms was made in the following manner. From each osmium fixed cochlea used for surface preparation a representative segment from each coil was examined. These samples usually [illegible] third of the length of a coil, were taken from the middle of the tympanic [illegible] f the cochlea (Fig. 1). The origin of these samples was noted so that a specimen from the basal coil was said to originate "1/2 coil" from the most basal part of the cochlea [illegible] near the oval window, a specimen from the second coil was "1 1/2 coil" from [illegible] another one from the third coil was called "2 1/2 coils from base", and a specimen from the apical coil was said to originate "3 1/2" coils from

base" The reason for using such terms is that the author wishes to emphasize that each specimen originates not only from a certain coil, but from a certain part of the coil, which represents the same location in every cochlea This standardization can be of crucial importance when comparing results of several studies in one laboratory or the results from different laboratories

For technical reasons we have chosen to register a distance containing three rows each containing 58 outer hair cells in each specimen This corresponds to roughly 0 5 mm length of the organ of Corti In general we have used the central part of the specimen for mapping The corresponding number of inner hair cells is not absolutely constant A slight variation can be found within a given cochlea and also between different cochleas This variance problem will be considered by *Engstrom, Ades,* and *Andersson* In the present material the average figure is 48 inner hair cells corresponding to 58 outer hair cells in one row, but the range of variation has been from 43 to 53 This means that each region included in the cochleogram contains more than 200 sensory cells, exactly plotted in their natural interrelation Usually each cochleogram includes four separate samples and the complete diagram thus includes almost 900 sensory cells, which is considered a fair, standard sampling of the cellular damage Spoiling of occasional parts of some specimens during preparation is difficult to avoid entirely Such damage can be minimized by care practice, and avoidance of undue haste by the operator To compensate for such damage in preparation, we have been obliged occasionally, to exclude some portion of a specimen to count the cells over a lesser distance than usual, or to take an adjacent portion for study

## 3 INNERVATION OF THE ORGAN OF CORTI

The complex innervation of the organ of Corti has engaged the attention of histologists for many decades Excellent descriptions of it based on earlier histologic methods have been given by, among others *Lorente de No* (1937), *Polyak* (1946), and *Fernandez* (1951) The present osmium tetroxide — zinc iodide staining combined with modern preparation techniques has given us much additional information With this staining method both nerve fibers and nerve endings within the organ of Corti are stained black and can easily be studied in surface specimens or sections with light microscopy In the following paragraphs a short description of the innervation of the guinea pig organ of Corti is given For details of innervation the reader is referred to the monograph by *Engstrom, Ades* and *Andersson*

From the spiral ganglion a large number of radial nerve fibers pass peripherally through the lamina spiralis ossea to the organ of Corti, penetrate the basilar membrane l[illegible]ng their myelin sheaths as they do so, and reach the inner spiral bundle In Figure 5 showing a surface specimen from the third coil of the cochlea the inner spiral bundle (ISB) is seen as an intense black broad ribbon in the upper part of the picture It runs spirally under the row of inner hair cells and consists of a large number of nerve fibers and the nerve endings attached to the base of the inner hair cells In a radial section of the organ of Corti (Fig 6) it is seen as a large accumu

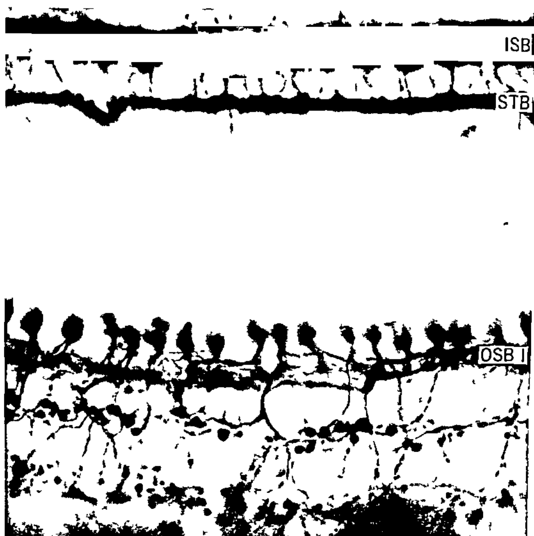



FIG 5 — *Surface preparation from the third coil of guinea pig cochlea The nerve endings form three different rows under the outer hair cells and it is seen how outer radiating fibers interconnect the three rows The nerve endings are very large under the first row of hair cells smaller under the second and very small under the third row Under the nerve endings of the first outer row the first outer spiral bundle (OSB I) can be seen indistinctly ISB—inner spiral bundle STB — spiral tunnel bundle Nerve staining 830 ×*

lation of neural material under an inner hair cell From the inner spiral bundle inner radiating fibers pass to the spiral tunnel bundle (STB) which follows a spiral course on the [illegible]r of the tunnel of Corti beneath the bases of the inner pillars From the spiral tunnel bundle radiating tunnel fibers cross the tunnel of Corti and reach the three outer spiral bundles (Figs 5 and 6) which run spirally under the corresponding rows of outer hair cells Some radial fibers connect the inner and outer spiral bundles directly [illegible] below the spiral tunnel bundle




FIG 6 — *Radial section of guinea pig organ of Corti from second coil of an acrylate embedded cochlea ISB — inner spiral bundle STB — spiral tunnel bundle OSB I, OSB II OSB III = the three outer spiral bundles with nerve endings under corresponding rows of outer hair cells Nerve staining 1000 ×*




[illegible] *from the apical coil of guinea pig cochlea showing a detail [illegible] the clusters of nerve endings innervating the first row of outer [illegible] cells with their nuclei are seen in profile in the background Note [illegible] course of the fibers to the individual sensory cells and also the distinct outer (OSB I) Nerve staining 1000 ×*

From the outer spiral bundles nerve fibers bend upwards and terminate in nerve endings under outer hair cells In the upper half of the cochlea as in Figures 5 and 7 the outer hair cells of the first row are supplied with large clusters of nerve endings whereas those of the second and third rows have nerve endings of considerably smaller volume Figure 7 shows a detail picture of the tulip like clusters of nerve endings of the outer hair cells of the first row The hair cells are seen in faintly out lined profile each hair cell having its own tulip of nerve endings attached to the base of the cell In the lower half of the cochlea the arrangement of the nerve endings of the outer hair cells differs considerably from that in the upper half In the basal coil all outer hair cells are supplied with large clusters of nerve endings and in the second coil the first two rows of outer hair cells have large endings but the outer most row is supplied by nerve endings of smaller volume

Electron microscopic studies by *Engstrom* (1958) and by *Smith* and *Sjostrand* (1961) have disclosed two types of nerve endings under outer hair cells one large and richly granulated the other smaller and sparsely granulated (Fig 8) All outer hair cells are supplied with small endings NE I in Figure 8 *Smith* and *Sjostrand* found large richly granulated endings under all outer hair cells in the basal coil under those of the first and second rows in the second coil and under those of the first row in the third and apical coils This distribution corresponds exactly to the distribution of the large clusters of nerve endings as revealed by light microscopic examination of surface specimens stained by the present technique Electron microscopic examination of sections stained with our nerve stain shows that the stain has a selective affinity for the large endings the small endings taking much less stain Corresponding differences were found in the staining properties of nerve fibers Although there is a striking difference in the electron density of the intensely stained and less stained neural matter both of them appear black under light microscopy *Engstrom* and *Fernandez* (1961) presented conclusive evidence that the large endings under outer hair cells represent terminations of the fibers of the efferent olivo cochlear bundle of Rasmussen They found that the large endings selectively degenerated after cross sectioning of the crossed olivo cochlear bundle in the mid brain Their results are supported by findings reported by *Spoendlin* and *Gacek* (1963)

The present staining technique has also given additional information on the arrangement of certain nerve fibers within the organ of Corti especially the course of the so called spironeurons which were described by *Retzius* *Lorente de No* and *Fernandez* This problem will be further discussed in connection with the description of the pattern of neural damage in the organ of Corti

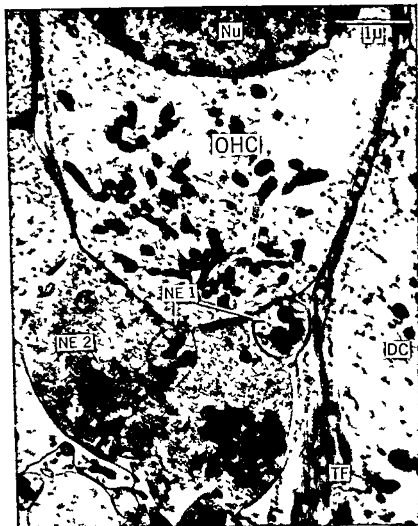



FIG 8 — *Electron n icrograph from tl e base of an outer hair cell (OHC) showing two types of nerve end ngs Nu — nucleus NE 1 = s uall sparsely granulated end ngs NE 2 = large ricl ly gran ilated endings TF — tonof brils in Deiters cell (DC) Epoxy resin 18600 ×*

# V RESULTS

## A. General survey of the development of hearing loss and cochlear damage in the experimental animals

### 1 NEOMYCIN

Thirty-one guinea pigs were treated with neomycin (Table I). The daily subcutaneous dose for all of them was 200 mg/kg body weight, the number of consecutive daily doses varying from four to eight.

TABLE I
*Animals treated with neomycin*

| Number of animals | Daily dose mg/kg body weight | Number of daily doses | Number of cochleas examined | | | |
|---|---|---|---|---|---|---|
| | | | As surface specimens | | As sections | |
| | | | Osmium fixation | Nerve staining | Osmium fixation | Nerve staining |
| 3 | 200 | 4 | 2 | 1 | 2 | 1 |
| 14 | 200 | 6 | 9 | 8 | 6 | 5 |
| 5 | 200 | 7 | 5 | 5 | 2 | 2 |
| 9 | 200 | 8 | 6 | 6 | 5 | 5 |

Three animals received a dose of 200 mg/kg body weight for four consecutive days. These animals were sacrificed within two days after the last injection. This dosage caused neither change in the threshold of the pinna reflex nor any histologic changes in the organ of Corti.

Fourteen animals were treated with 200 mg/kg body weight for six consecutive days. They were allowed to survive for different lengths of time, so that the first animal was sacrificed on the day of the last injection and the last animal 40 days after the treatment was stopped. Three of the animals of this series lost the pinna reflex within a week after the end of the treatment and a fourth showed a considerably higher threshold. Most of them, however, showed no hearing loss detectable by this rough testing method. Neither was any damage found upon histologic examina- [illegible] chleas of the animals except for scattered loss of outer hair cells. One [illegible] deafened on the second day and sacrificed on the 16th day after the treatment [illegible] ception. Histologic study of the cochleas revealed almost complete [illegible] cells. In other deafened animals no histologic changes were found in th[illegible] greater than those from animals with preserved pinna reflex.

Five animals received a dose of 200 mg/kg body weight for seven consecutive days. In order to study the late effect of the antibiotic these animals were allowed to survive for relatively long times, 26 to 84 days after the end of the treatment. Two of them lost the pinna reflex within a week after the last injection, and all were deaf prior to death. Correspondingly, histologic studies revealed pronounced degeneration of both sensory cells and neural elements following the pattern described below. Complete loss of sensory elements of the organ of Corti was found in no animal of this series.

Nine animals received 200 mg neomycin per kg body weight for eight consecutive days. With the exception of two animals with the shortest survival times, they appeared to be deaf shortly after the last injection, most of them within three days. The first animal was sacrificed three days after the final injection, the others at successive intervals with the last at 33 days. Histologically, a wide range of degeneration patterns of the organ of Corti was recorded, from slight changes in the first animals to extensive damage in the animal with the longest survival time. The given dose seemed sufficient to result in complete loss of sensory elements of the organ of Corti, given a long enough period to complete its effect.

## 2. KANAMYCIN

The group treated with kanamycin numbered 13 animals (Table II). Two animals received 400 mg kanamycin per kg body weight for seven consecutive days. They survived 33 and 67 days respectively after the treatment. In the first one the pinna reflex was abolished within four days after the last injection, while in the latter one, it was unchanged at decapitation. Correspondingly, severe damage was found in the cochleas of the former animal, but only slight changes in the latter one.

TABLE II
*Animals treated with kanamycin*

| Number of animals | Daily dose mg/kg body weight | Number of daily doses | Number of cochleas examined | | | |
|---|---|---|---|---|---|---|
| | | | As surface specimens | | As sections | |
| | | | Osmium fixation | Nerve staining | Osmium fixation | Nerve staining |
| 2 | 400 | 7 | 2 | 1 | 1 | — |
| 3 | 400 | 8 | 3 | 3 | — | — |
| 8 | 400 | 10 | 3 | 5 | 4 | 4 |

Three animals were given 400 mg kanamycin per kg body weight for eight consecutive days. All of these lost the pinna reflex on the day following the administration of the last dose. They were sacrificed 6, 8, and 14 days respectively after treatment. Histologic examination of the cochleas revealed in all animals considerable damage to the outer and inner hair cells and to the neural elements of the organ of Corti following the same pattern as that seen in neomycin treated animals.

Eight animals received 400 mg kanamycin per kg body weight for 10 consecutive days In all animals of this series the pinna reflex disappeared between the seventh and tenth day of treatment On the basis of histologic study it was evident that this dosage of the drug rapidly leads to complete destruction of the sensory cells with subsequent neural damage In one animal only, sacrificed on the day of the last injection, was it possible to record the cellular loss on the standard cochleogram In the others, surviving 4 to 28 days after the end of the treatment, destruction of the organ of Corti was so devastating as to make this type of recording impossible

## 5 FRAMYCETIN

Fourteen animals were treated with framycetin (Table III) The five animals of the first series were given different doses of the drug in order to define its degree of ototoxicity in the guinea pig Unlike the other series the animals of this series were not injected on consecutive days, the treatment being interrupted twice for two day periods One of the animals received 11 daily doses of 50 mg framycetin per kg body weight, a second one 11 doses of 100 mg/kg body weight, no hearing loss and no histologic damage resulting in either One of the animals was given eight injections of 200 mg/kg body weight and two animals were given ten injections of 200 mg/kg body weight These three animals lost the pinna reflex within four days after the last injection They were sacrificed during the second week after treatment Histologic examination revealed considerable loss of sensory cells and degeneration of neural elements

TABLE III
*Animals treated with framycetin*

| Number of animals | Daily dose mg/kg body weight | Number of daily doses | Number of cochleas examined | | | |
|---|---|---|---|---|---|---|
| | | | As surface specimens | | As sections | |
| | | | Osmium fixation | Nerve staining | Osmium fixation | Nerve staining |
| 1 | 50 | 11 | 1 | 1 | – | – |
| 1 | 100 | 11 | 1 | 1 | – | – |
| 5 | 200 | 8 | 8 | 1 | 1 | – |
| 5 | 200 | 9 | 5 | 5 | – | – |
| 2 | 200 | 10 | 2 | – | 2 | – |

A series of four animals was given 200 mg framycetin per kg body weight for eight consecutive days resulting in a loss of the pinna reflex within a week after the last injection in all animals At histologic examination cochlear damage of the same type and degree was found as in the three animals of the first framycetin series treated with the same daily dose

Five animals were given 200 mg framycetin per kg body weight for nine consecutive days The pinna reflex disappeared in all five cases between the seventh and tenth days of treatment Histologic examination revealed pronounced damage in the cochleas This dosage seemed to result in rapid and complete loss of sensory cells and severe neural degeneration as was the case with the largest dosage of kanamycin The animals of this series were allowed to survive from 2 to 29 days after the end of the treatment

## B Morphology in the degeneration of individual sensory cells

In connection with the description of the surface specimen technique it was mentioned that the main virtue of the method is that it provides the examiner with a panoramic view of the whole structural pattern of the organ of Corti It should also be noted that morphologic changes in individual sensory cells can be studied in surface specimens with as great accuracy as in ordinary sections of the cochlea

Nuclear changes in outer hair cells are usually the first pathologic phenomena observed in cochleas of animals treated with ototoxic antibiotics In early stages of degeneration the nuclei undergo considerable swelling In later stages they finally break down and the chromatin is dispersed in the cytoplasm Rests of chromatin are regularly found in the cytoplasm of degenerating cells in specimens with advanced damage The nuclear changes involve the inner hair cells also and in a similar way but in this study special attention was not paid to the temporal relations of nuclear changes between outer and inner hair cells or between different rows of outer hair cells Generally the findings with respect to sensory cell nuclei in the present experiments were in agreement with those reported by *Neumann* and *Neubert* (1958) and by *Beck* and *Krahl* (1962) in guinea pigs treated with ototoxic antibiotics

A second early change which is often observed is a disarrangement of the pattern of the sensory hairs on individual outer hair cells In normal specimens (Fig 2) the W formed by the hairs always shows a uniform conformation but in degenerating cells the W figure becomes an irregular line the hairs often pointing in different directions This may be due to softening of the normally dense cuticular plates of the cells allowing movement of the rootlets of the individual hairs in the cuticle The cuticular surface of the last remaining sensory cells in a damaged row or area often has an abnormally heterogeneous appearance apparently as a result of degeneration (Figs 9 and 10) The disarrangement of hairs is not seen in all specimens with cellular damage and complete loss of hairs is not characteristic of early stages of degeneration some remaining hairs often being found on severely degenerated cells The disarrangement of the hairs has been reported previously by *Hawkins* and *Engström* (1964)

The most characteristic feature of cell degeneration as seen in surface specimens is the collapse of the sensory cells resulting in typical figures which we have called collapsed cells In normal specimens each sensory cell seems to have a certain number of attachments to the neighboring structures This is most clearly seen

among the outer hair cells of the second row, e g in Figures 3 and 4, where each of them is connected to four adjacent sensory cells two in the first row and two in the third row These attachments evidently correspond to the desmosomes or attachment plaques that have been observed by both light and electron microscopy in other epithelial cells The site and number of these attachments seem to determine to a high degree the appearance of a 'collapsed cell in a surface specimen

During its degeneration the volume of the outer hair cell diminishes and the walls of the cell become detached from the surrounding phalanges of the Deiters' cells, except at the above mentioned attachment zones Thus a 'gap', probably a fluid filled space, appears between the collapsing sensory cell and the surrounding phalanges Gradually the cytoplasm of the degenerating sensory cell diminishes finally to disappear completely and a characteristic figure formed by the walls of the collapsed cell remains the space between this slender, membranous figure and the walls of the surrounding phalanges still existing The collapsed cells are usually most distinctly seen in the second row of outer hair cells, as seen in Figures 11 and 13 The collapsed cells of the third row are similar to those of the second row, but in the first row, the collapse figure has a somewhat different appearance (Fig 13) The degeneration of individual inner hair cells follows the same principles (Figs 13—15), but the contours of collapsed cells are often less distinct, especially in the upper parts of the cochlea where the volume of the cells is small

A second type of the collapsed cell can be found among the outer hair cells of the second and third rows (Fig 9) Here the surrounding phalanges of the Deiters cells have completely filled up the space around the suspender like figure This type of collapsed cell is found as a late result of relatively slight damage from antibiotics when only scattered sensory cells have been lost and the surrounding supporting cells spared A few such collapsed cells can also be found in specimens from normal animals as a result of earlier traumatic degeneration or perhaps of congenitally missing cells Thus for example the collapsed cells in the second and third rows of outer hair cells in Figure 9 cannot with certainty be attributed to damage from antibiotics In practical use this state of things does not create any problems, as this type of collapsed cells is not common in systematic degeneration

## C. Pattern of cellular damage in the organ of Corti

Examination of the surface specimen material of the present study combined with systematic recording of the damage by means of cochleograms resulted in a concept of the pattern of sensory cell degeneration This pattern was found to follow the same general principles with each of three drugs administered In the following paragraphs typical micrographs illustrating different stages of cochlear degeneration will be described and then the concept of the progressive degeneration pattern will be presented by me[illegible]s of cochleograms

Figure 9 shows a specimen from the third coil of the cochlea of a guinea pig of the framycetin series Most of the outer hair cells of the first row have collapsed while the four remaining ones stand out distinctly The two collapsed cells, one in the second and one in the third row, were discussed in the previous chapter




FIG 9 – *Guinea p g Su 253 R framycet n 200 mg kg body weight for 8 consecutive days sacrificed 11 days after last injection Surfa e preparation 2½ coils from base of cochlea The first row of outer hair cells 1) has been severely damaged and the individual cells are replaced by coll pse f gure arrow ex ept for four remaining cells In the second (2) and third (3) rows in th r t p llapse figure (arrows) is seen one in each row This type is often foun l l r al p ns indicating congenitally missing cells or cells degener ated because t y l l e rl r damage Phase contrast 1640 ×*

Figure 10 shows another field in the same specimen where all outer hair cells of the *second and third rows have a normal appearance but most of those of the first row* have been destroyed In the upper part of the picture the row of inner hair cells normal in appearance, is indistinctly seen being somewhat out of focus

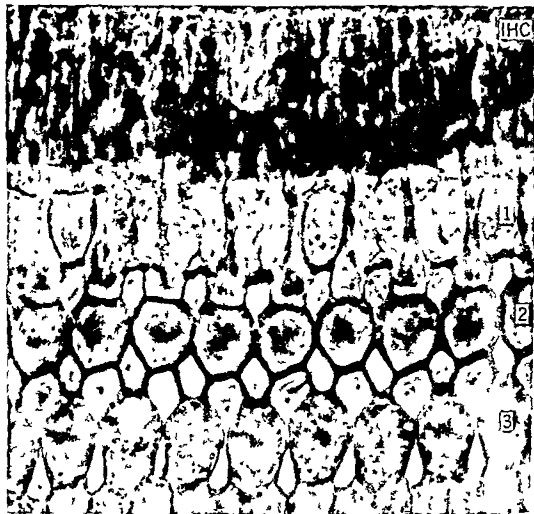



FIG 10 — *Guinea pig Su 253 R framycetin 200 mg/kg body weight for 8 consecutive days sacrificed 11 days after last injection Surface preparation 2 1/2 coils from base of cochlea The first row of outer hair cells (1) has been severely damaged Three cells in this row remain with partly damaged cuticular plates The cells of the second (2) and third (3) rows have a normal appearance A row of inner hair cells (IHC) is seen faintly in the upper part of the picture slightly out of focus Phase contrast 1640 ×*

The micrograph in Figure 11 was taken from a surface preparation of the third cochlear coil of a guinea pig treated with neomycin The only remaining outer hair cell in the first row stands out distinctly among its degenerated neighbors while all cells in the second row and several in the third row have collapsed This picture also shows beginning breakdown of the supporting structures In the area between the arrows the regular collapse figures have been transformed to an irregular membranous network




FIG 11 — *Guinea pig Su 238 L neomycin 200 mg/kg body weight for 7 consecutive days, sacrificed 68 days after last injection Surface preparation 2½ coils from base of cochlea Severe destruction of outer hair cells One outer hair cell has remained in the first row and six in the third row Note distinct collapse figures in second row (white arrow) and the beginning [illegible] of supporting framework of the third and second rows (between black arrows) Pl[illegible]*

Figure 12 shows complete degeneration of all outer hair cells of the basal coil in an animal treated with framycetin In the upper part of the picture can be seen the row of inner hair cells all of which appear to be intact




FIG 12 — *Guinea pig Su 253 R framycetin 200 mg/kg body weight for 8 consecutive days sacrificed 11 days after last injection Surface preparation ½ coil from base of cochlea All outer hair cells in the three rows have degenerated The row of intact inner hair cells (IHC) is seen distinctly above the ribbon of the pillar heads (P) Phase contrast 1970* ×

Kanamycin damage to both outer and inner hair cells is illustrated by Figure 15 which shows a specimen from the third coil of the cochlea. Among the outer hair cells the first row is completely destroyed while two cells in the second row and all of those of the third row have been spared. Among the inner hair cells only two remain while all the others have collapsed.

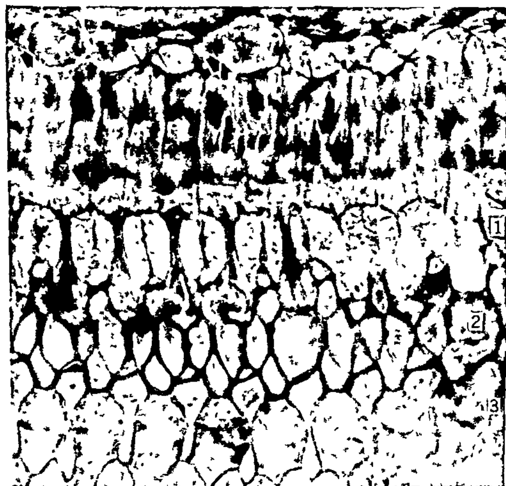



*FIG. 15. — Guinea pig S. 271 L. Kanamycin 400 mg/kg body weight for 8 consecutive days, sacrificed 14 days after last injection. Surface preparation 2½ coils from base of cochlea. Damage to both outer and inner hair cells is seen in this specimen. The two remaining inner hair cells (arrows) stand out distinctly. The third row of outer hair cells remains as well as two cells in the second row. Phase contrast. 1070 ×.*

Figure 14 shows a specimen from the third coil of the cochlea of an animal treated with neomycin The degree of hair cell damage is similar to that seen in Figure 13 The normal and collapsed outer hair cells can be easily distinguished from each other A single, normal appearing inner hair cell is seen, the sensory hairs on the surface being discernible Beginning breakdown of supporting structures of outer hair cells is seen in the area marked by the arrows where two cells of the first row, together with the separating phalanx of a Deiters' cell, have become almost homogeneous in appearance

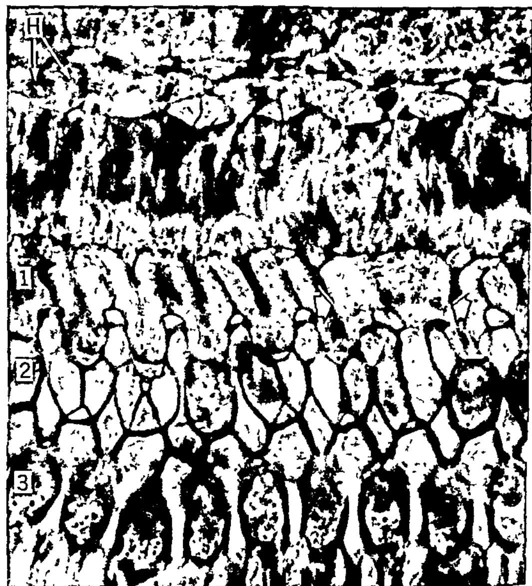



*FIG 14 — Guinea pig Su 204 L neomycin 200 mg/kg body weight for 8 consecutive days sacrificed [illegible] after last injection Surface preparation 2½ coils from base of cochlea Only one inner [illegible] cell has been preserved from degeneration Hairs (H) are seen on its cuticular surf[illegible] rd row of outer hair cells and a few cells in the second row also remain Note [illegible] akdown of the Deiters cells of the first and second rows (between arrows) Phase*

Figure 15 a specimen from the kanamycin series shows a general view of the third coil of the organ of Corti In the third row of outer hair cells beginning break down of the supporting structures is seen To the right there is a gap in the row of the inner pillar heads indicating two degenerated inner pillars The accompanying cochleogram has been reconstructed from the micrograph in order to visualize the method of graphic registering used in this study By comparing it with the micro graph it can be seen that each cell with normal appearance has been registered as an open circle (O) and each degenerated cell as a solid circle (●)

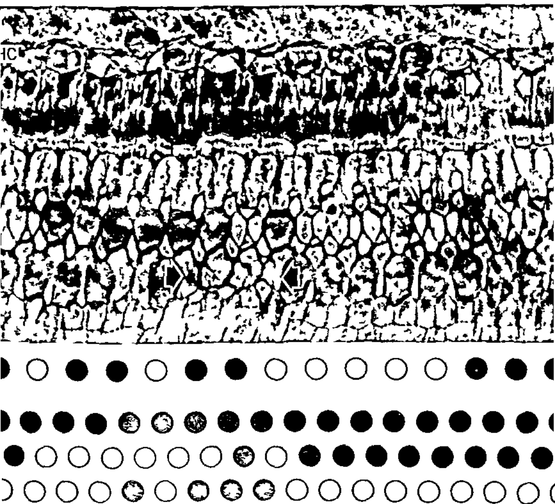


FIG 15 — Gu \ I I k na n 400 mg kg body weight for 8 consecutive days sacrificed 14 d S face preparation 2½ coils from base of cochlea All outer hair cell l en lost and the second row has been severely damaged but most of t i re ia n Note the gap in the ribbon of the pillar heads (P) and cat n r pillars (white arrows) and the beginning breakdown of the supporting fra e o k have also a ed When c a g l l as an pen ir le and

Figure 16 a micrograph from a second coil specimen shows complete loss of outer hair cells as a result of administration of kanamycin The collapsed cells of the first and second rows with the surrounding phalanges in place are seen distinctly In place of the third row of collapsed outer hair cells, structureless material in an irregular membranous network is found as a result of the breakdown of the supporting Deiters cells

Figure 17 shows severe damage to the basal coil of the cochlea from kanamycin in a radial section of the organ of Corti The outer hair cells with the supporting Deiters' cells have been replaced by structureless material The contours of a partly collapsed inner hair cell are faintly seen

The first scattered losses of outer hair cells found after relatively small doses of antibiotics or in early stages of degeneration after large doses are shown by a cochleogram from an animal treated with neomycin (Fig 18) The collapsed cells are unevenly distributed in different coils and different rows of the organ of Corti, no definite pattern in the distribution being apparent




*FIG ... 0 I kanamycin 400 mg kg body weight for 8 consecutive days sacrifice ... ection Surface preparation 1½ coils from base of cochlea Complete ... The collapsed cells of the first (1) and second (2) rows are seen ... ... neration of the supporting framework has transformed the third row into ... Phase contrast 1000*

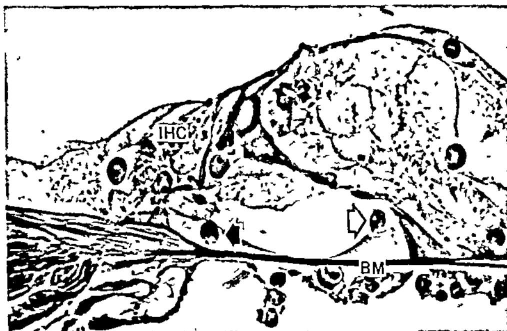



FIG 17 — *Guinea pig Su 211 R kanamycin 400 mg/kg body weight for 10 consecutive days sacrificed 4 days after last injection Radial section from basal coil of epoxy resin embedded cochlea Severe destruction of outer hair cells and Deiters cells The contours of a partly collapsed inner hair cell (IHC) are just visible The outer pillar is seen somewhat distorted with the nucleus at its base (white arrow) as well as the nucleus of the inner pillar (black arrow) BM — basilar membrane Phase contrast 910 ×*

SW 194 L NEOMYCIN 200 MG/KG BODY WEIGHT FOR 6 CONSECUTIVE DAYS SACRIFICED 6 DAYS AFTER LAST DOSE

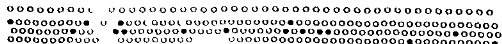
3 1/2 coils from base of cochlea



2 1/2



1 1/2



1/2

FIG 18 — *This cochleogram shows scattered outer hair cell loss after a relatively small dose of the antibiotic No definite pattern can be seen in the distribution of missing cells*

The first signs of a systematic loss of outer hair cells are shown by a cochleogram from an animal treated with framycetin (Fig 19) The first row of outer hair cells in the basal coil has undergone considerable degeneration and several collapsed cells are found also in the two outer rows of the same coil Only a few scattered cells are missing in the upper parts of the cochlea

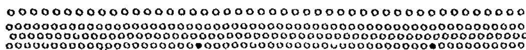
At the next stage of degeneration almost all outer hair cells of the basal coil are destroyed Two cochleograms demonstrate this stage In the first (Fig 20), from an animal treated with framycetin practically no damage can be seen in the upper coils of the cochlea In the second (Fig 21) from an animal treated with neomycin, the largest number of collapsed cells in the upper parts of the cochlea are found in the first rows of the apical and third coils

From here on a complete loss of outer hair cells in the basal coil is seen in the following cochleograms which illustrate more advanced stages of degeneration Figure 22 shows a cochleogram from the kanamycin series in which, in addition to the damage in the basal coil the two innermost rows of outer hair cells in the second coil are very severely affected

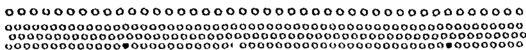
A cochleogram from an animal given framycetin (Fig 23) illustrates the same stage of outer hair cell loss The pattern of the degeneration is the same in the lower half of the cochlea and in the upper half the cells of the first row in both the third and apical coils show a tendency to systematic loss

SW 253 R FRAMYCETIN 200 MG/KG BODY WEIGHT FOR 8 CONSECUTIVE DAYS
SACRIFICED 20 DAYS AFTER LAST DOSE

3 1/2 coils from base of cochlea



2 1/2



1 1/2



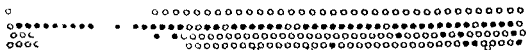


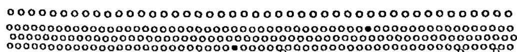
FIG 1? ... ates beginning tendency toward a systematic loss of outer hair cells ... has been severely damaged

SW 254 L FRAMYCETIN 200 MG/KG BODY WEIGHT FOR 8 CONSECUTIVE DAYS SACRIFICED 15 DAYS AFTER LAST DOSE

3 1/2 coils from base of cochlea



2 1/2



1 1/2



1/2



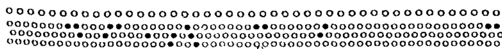
FIG 20 — *The cochleogram shows an almost complete loss of outer hair cells in the basal coil The three upper coils of the cochlea are practically undamaged*

SW 228 L NEOMYCIN 200 MG/KG BODY WEIGHT FOR 7 CONSECUTIVE DAYS SACRIFICED 26 DAYS AFTER LAST DOSE

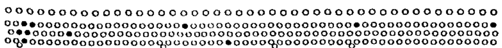
3 1/2 coils from base of cochlea



2 1/2



1 1/2



1/2

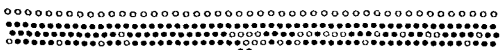
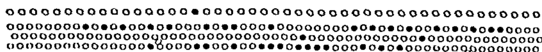


FIG 21 — *Almost all outer hair cells in the basal coil have degenerated in this cochlea In the upper two coils there is a slight tendency to systematic degeneration in the first row of outer hair cells*

SW 208 L KANAMYCIN 400 MG/KG BODY WEIGHT FOR 10 CONSECUTIVE DAYS SACRIFICED AT THE DAY OF LAST DOSE

3 1/2 coils from base of cochlea



2 1/2



1 1/2



1/2

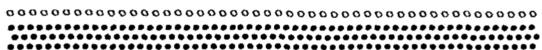


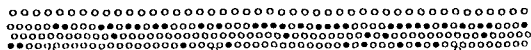
FIG 22 — *This cochleogram illustrates a complete loss of outer hair cells in the basal coil and severe damage to those in the first two rows of the second coil In the two upper coils the largest number of "collapsed" outer hair cells is found in the first row One inner hair cell is missing in the apical coil*

SW 255 L FRAMYCETIN 200 MG/KG BODY WEIGHT FOR 8 CONSECUTIVE DAYS SACRIFICED 11 DAYS AFTER LAST DOSE

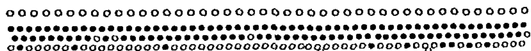
3 1/2 coils from base of cochlea



2 1/2



1 1/2



1/2

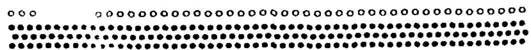


FIG 23 ... *a complete loss of outer hair cells is seen in the basal coil and sub total damage ... rows of the second coil In the two upper coils the largest number of damaged ... is seen in the first row Three inner hair cells are missing in the apical c...*

The next two cochleograms taken from the neomycin series (Figs 24 and 25), illustrate a very characteristic stage in the pattern of outer hair cell loss All three rows in the basal coil the first two rows in the second coil and the innermost row in the third and apical coils are almost completely missing In the latter two coils, almost no damage is found in the second and third rows of outer hair cells

Compared with these two cochleograms that from an animal of the kanamycin series (Fig 26) has the same general pattern of outer hair cell loss with somewhat more advanced degeneration in the third row of the second coil but less damage in the first row in the upper parts of the cochlea

Two cochleograms (Figs 27 and 28) from animals of the kanamycin series represent the most advanced stages of cellular damage that could be graphically registered as cochleograms In both of these cochleas also the third row of outer hair cells in the second coil has been completely lost while the two outer rows in the upper parts of the cochlea have been mostly spared

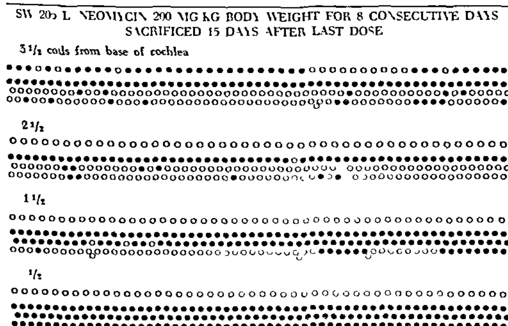



FIG 24 — *This cochleogram illustrates a typical pattern of sensory cell degeneration from neomycin Note the different pattern of outer hair cell loss in different coils they have been damaged in all three rows of the basal coil in the first two rows of the second coil and in the first row of the two upper coils A large number of inner hair cells in the apical coil have been damaged but missing inner hair cells are not seen in the other coils*

SW 204 L NEOMYCIN 200 MG/KG BODY WEIGHT FOR 8 CONSECUTIVE DAYS
SACRIFICED 10 DAYS AFTER LAST DOSE

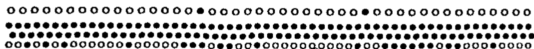
3 1/2 coils from base of cochlea



2 1/2



1 1/2



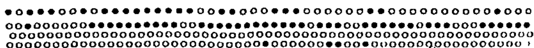
1/2



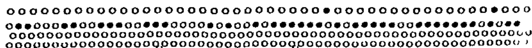
FIG 25 — *In this cochleogram the pattern of outer hair cell degeneration is the same as in Figure 24 Note the advanced inner hair cell damage in the two upper coils, while the majority of outer hair cells of the second and third rows remain undamaged*

SW 218 L KANAMYCIN 400 MG/KG BODY WEIGHT FOR 8 CONSECUTIVE DAYS
SACRIFICED 6 DAYS AFTER LAST DOSE

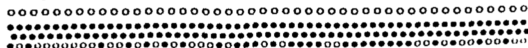
3 1/2 coils from base of cochlea



2 1/2



1 1/2



1/2



FIG 26 — *Severe damage to outer hair cells in the two lower coils of the cochlea Only the outermost row in the second coil is partly preserved In the two upper coils the innermost row has also been severely damaged Observe beginning inner hair cell loss at apex of cochlea*

SW 221 L KANAMYCIN 400 MG/KG BODY WEIGHT FOR 8 CONSECUTIVE DAYS SACRIFICED 14 DAYS AFTER LAST DOSE

3½ coils from base of cochlea



2½



1½



½



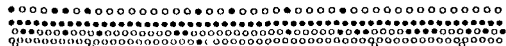
FIG 27 — *This cochleogram illustrates advanced sensory cell degeneration from kanamycin In the basal and second coils all outer hair cells have been lost, but in the two upper coils only the first row has degenerated completely Observe the pattern of inner hair cell degeneration with decreasing damage from the apex toward the base*

SW 220 L KANAMYCIN 400 MG/KG BODY WEIGHT FOR 8 CONSECUTIVE DAYS SACRIFICED 8 DAYS AFTER LAST DOSE

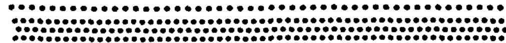
3½ coils from base of cochlea



2½



1½



½

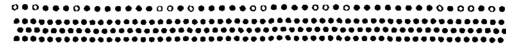


FIG 28 — *This cochleogram shows advanced sensory cell degeneration as a result of administration of kanamycin Complete loss of outer hair cells in the two lower coils and of those in the first row of the upper coil In this cochlea the pattern of inner hair cell degeneration is somewhat different from that usually found, as the inner hair cells here have been more severely affected in the two lower coils than in the third coil Usually the damage to inner hair cells decreases evenly from the apex towards the base of the cochlea*

The degeneration of the inner hair cells also follows a definite pattern which is in a sense opposite to that of the outer hair cells The pattern can be discerned in the same series of cochleograms In Figure 23 three collapsed inner hair cells are found in the apical coil In the next cochleogram of the series (Fig 24) the greater number of the inner hair cells in the apical coil have undergone degeneration whereas those in the lower parts of the cochlea still have a normal appearance In Figure 25 the loss of inner hair cells has begun from the apex and reached the third coil but only two missing inner hair cells are found in the lower half of the cochlea The same pattern of inner hair cell degeneration resulting from kanamycin is seen in the cochleograms in Figures 26 and 27 In the former the degeneration affects mainly the top coil in the latter collapsed inner hair cells are found in all coils of the cochlea their number decreasing towards the base Figure 28 shows a somewhat deviant pattern of inner hair cell loss from kanamycin the damage being greater in the second and basal coils than in the third coil

The degeneration of inner hair cells thus usually begins at the apex of the cochlea and proceeds towards the base In the present material the inner hair cells of the two apical coils usually had undergone degeneration at a stage in which the majority of outer hair cells in the outer two rows of the same coils had so far been spared

From the most advanced stages of degeneration seen in the series of cochleograms the degeneration rapidly progresses to a complete loss of all sensory cells During these late stages of cellular damage considerable breakdown of the supporting structures also takes place and makes it impossible to construct reliable cochleograms however it is usually still possible to recognize to which row the last remaining cells belong They were usually found among the outer hair cells in the second and third rows of the apical coil and among the inner hair cells of the basal coil

It must be pointed out that a certain degree of liberty has been taken in constructing the cochleograms representing stages of degeneration in which there has been complete loss of outer hair cells For example in the cochleas from which Figures 22–28 are derived no single outer hair cell of normal appearance was seen in the basal coil specimens moreover the concomitant breakdown of supporting structures so disorganizes the appearance of the outer hair cell region that is impossible to identify each and every degenerated hair cell and assign it to its proper row For this reason the outer hair cell region of the basal coil in such cochleograms had to be reconstructed somewhat schematically from knowledge of the normal structural pattern This liberty seems to be justified as a means of portraying the comparative condition of all coils simultaneously because where all the cells are destroyed it cannot matter exactly how many there were before destruction The only possibility of error (a very minor one) lies in the fact that if there had been supernumerary cells or slight irregularities of pattern these being unidentifiable would be omitted from the reconstruction The same situation is foun[illegible] n the second coil of the cochleograms in Figures 27 and 28

The p[illegible] f hair cell loss following the administration of each of the three drugs was found [illegible] l w the same general principles as is evident in the series of cochleograms h w [illegible] inamycin seems to have a slightly greater affin ty for the second

and basal coils of the cochlea than do neomycin and framycetin the difference being more pronounced in the second coil This holds for both outer and inner hair cells and is seen in two cochleograms of the series In the cochleogram of Figure 26 about half of the outer hair cells of the third row in the second coil have undergone degeneration although several outer hair cells in the first row of the third and apical coils are still intact In the cochleogram of Figure 28 all inner hair cells of the second coil and most of those in the basal coil have been lost but only a few collapsed inner hair cells are found in the third coil In contrast evenly decreasing loss of inner hair cells from apex to base of the cochlea was always found among neomycin and framycetin treated animals

In the description of the micrographs showing different stages of sensory cell loss some attention was paid to the accompanying damage to supporting cells Generally it was evident that damage to the sensory cells always preceded deterioration of the supporting structures In most of the micrographs for example advanced degeneration of hair cells is found in the absence of any visible damage to the supporting elements Deiters cells seem to be the most sensitive of the supporting elements Following the degeneration of the sensory cells they have been supporting the Deiters cells coalesce to a structureless material together with degenerated sensory cells Initially this breakdown is localized involving no more than two or three phalangeal and adjacent outer hair cells (Fig 14) but progresses to include whole rows of Deiters cells after complete loss of the corresponding outer hair cells has taken place (Fig 16) The initially localized breakdown of the Deiters cells has no detectable site of predilection but may be found associated with outer hair cell damage in specimens from any coil of the cochlea At a late stage of degeneration all outer hair cells and Deiters cells coalesce to an almost homogeneous structureless mass (Fig 17)

As compared with the Deiters cells the pillar cells show considerable resistance to the toxic effects of the antibiotics Scattered loss of a few inner and outer pillars is usually associated with advanced degeneration of the inner hair cells while missing pillars are rarely found in specimens in which all inner hair cells have a normal appearance In surface specimens degeneration of individual pillars creates gaps in the otherwise regular ribbon of the pillar heads (Fig 15) Even in specimens with complete loss of sensory cells and severe destruction of other supporting cells most of the pillars still remain holding the organ of Corti upright In Figure 17 with severe damage to the organ of Corti the outer pillar is seen to be slightly distorted but the nuclei of both outer and inner pillars are clearly seen at the base of the cells and display a fairly normal appearance

## D Pattern of neural damage in the organ of Corti

The nerve staining technique described in an earlier chapter made it possible to study the degeneration of the nerve endings and nerve fibers within the organ of Corti parallel to the study of hair cell damage Even in nerve stained specimens it is always possible to get some idea of the condition of the sensory cells although it is more convenient to study sensory cell changes under phase contrast microscope after




FIG 29 — *Guinea pig Su 204 R neomycin 200 mg/kg body weight for 8 consecutive days sacrificed 10 days after last injection Surface preparation 3½ coils from base of cochlea The picture illustrates the 'wilting' of the large, tulip like nerve endings innervating the first row of outer hair cells Only two of the 'tulips' remain OSB I = first outer spiral bundle Compare with the corresponding normal picture (Fig 7) Nerve staining 1000* ×

fixation in osmium tetroxide alone In the present material the author usually had to make observations on neural damage in specimens with complete or severe loss of sensory cells as it was difficult to calibrate the total dosage to result in a moderate damage with the usual large daily doses A given number of daily injections might cause almost no damage at all while addition of one further dose resulted in very severe or complete loss of sensory cells

Under these circumstances i e short time treatment with large daily doses the degeneration of neural elements is clearly secondary to the damage to sensory cells Degeneration of nerve endings or nerve fibers was never found in specimens with intact sensory cells not even in animals which had survived one to two months after the last injection On the other hand a normal neural pattern was found even in several cochleas with relatively advanced loss of sensory cells

Although secondary to cellular damage early changes in the nerve endings appear within a few days after the loss of corresponding hair cells In surface specimens the

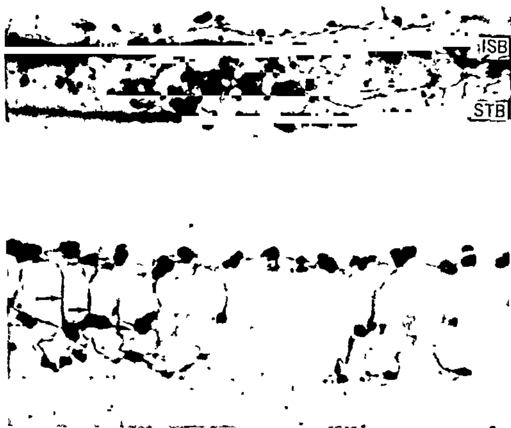



FIG 30 – *Guinea pig Su 213 L, kanamycin 400 mg/kg body weight for 10 consecutive days, sacrificed 4 days after last injection. Surface preparation 1½ coils from base of cochlea. Degeneration of nerve endings of outer hair cells. The outer radiating fibers (arrows) connecting the outer spiral bundles are distinctly seen at left. A gap has appeared in the neural pattern of the second and third rows. The inner spiral bundle (ISB) and the spiral tunnel bundle (STB) have a normal appearance. Nerve staining 980 ×*

first alterations are found in the large clusters of nerve endings innervating certain of the outer hair cells. In the upper parts of the cochlea, where the first row of outer hair cells is supplied by large endings (Figs. 5 and 7), a "wilting" of these tulip-like clusters of nerve endings is found, as they droop down to the corresponding outer spiral bundle and the "stalks" of the "tulips" disappear (Fig. 29). This is probably not due to their degeneration, but rather to collapse of the whole organ of Corti as a result of the destruction of the sensory cells. True degeneration of the nerve endings of the outer hair cells soon becomes evident, as they gradually diminish in size and lose their original shape, becoming irregular and disorganized (Fig. 30). Local disappearance of all nerve endings and nerve fibers in the outer spiral bundles is often seen, leaving gaps in the disintegrating neural pattern (Fig. 30).

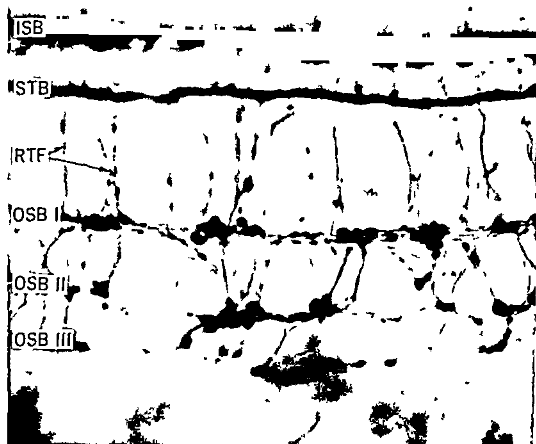



FIG. 51 — *Guinea pig Su 226 R, neomycin 200 mg/kg body weight for 8 consecutive days, sacrificed 35 days after last injection. Surface preparation 1½ coils from base of cochlea. Severe degeneration of outer nerve endings with coalescence of neural material. The course of individual spiral nerve fibers can be followed in the outer spiral bundles (OSB I, OSB II, OSB III). Individual nerve fibers are also distinctly seen in the radiating tunnel fibers (RTF). Slight reduction in size of the inner spiral bundle (ISB) and of the spiral tunnel bundle (STB). Nerve staining. 520 ×*

A typical phenomenon characteristic of advanced neural degeneration is shown by Figures 51 and 52. The disintegrating nerve endings and nerve fibers coalesce to form large deformed accumulations of neural material. In Figure 51 these accumulations are found in the outer spiral bundles and in Figure 52 one large accumulation is seen attached to the spiral tunnel bundle. This coalescence of neural detritus is *observed at a relatively late stage of degeneration, usually four to five weeks after* the loss of the corresponding hair cells. No details of the coalescing neural material can be discerned by light microscopy. As degeneration progresses the accumulations gradually diminish and finally disappear while a few nerve fibers still remain. The same coalescence is also visible in radial sections of the organ of Corti as seen in Figure 53. All hair cells in this specimen have been destroyed and the whole organ of

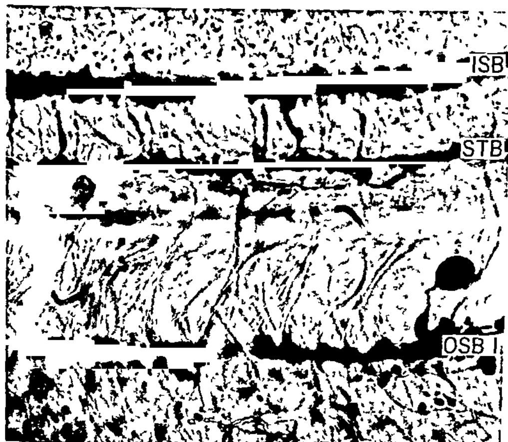



FIG 32 — *Guinea pig Su 2?3 L, kanamycin 400 mg/kg body weight for 10 consecutive days sacrificed 28 days after last injection Surface preparation 2½ coils from base of cochlea To the left is seen a large accumulation of degenerating neural material attached to the spiral tunnel bundle (STB) and to some radiating tunnel fibers At right a similar accumulation is seen attached to the first outer spiral bundle (OSB I) The large tulip like nerve endings of the first outer row have disappeared completely ISB = inner spiral bundle Nerve staining 820 ×*

Corti has undergone partial collapse Accumulations of coalescing neural material are seen in the outer spiral bundles

D_neration of the inner spiral bundle with its nerve endings and that of the spiral tunnel bundle becomes visible much later than that of the outer nerve endings and outer spiral bundles In Figure 31 where the outer spiral bundles have undergone considerable degeneration no more than slight changes can be seen in the inner bundles with slight reduction of size of the spiral tunnel bundle and beginning rarefaction of the inner spiral bundle With long survival times after large doses of antibiotics pronounced damage is found also in these bundles (Fig 34) The spiral tunnel bundle is usually better preserved than the inner spiral bundle as seen in the same micrograph In this specimen only one or two single nerve fibers were left from the

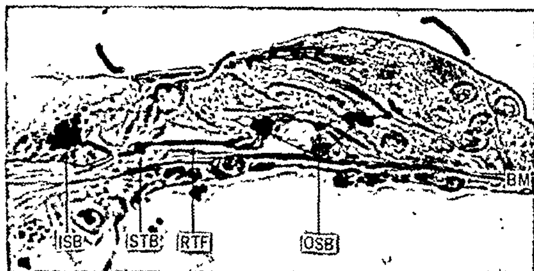



FIG 33 — *Guinea pig Su 223 R, kanamycin 400 mg/kg body weight for 10 consecutive days sacrificed 28 days after last injection Radial section from second coil of an acrylate embedded cochlea The sensory cells have been completely destroyed, and the whole organ of Corti partly collapsed Accumulations of degenerating neural material are seen at the outer spiral bundles (OSB) The inner spiral bundle (ISB) and the spiral tunnel bundle (STB) are relatively well preserved RTF = radiating tunnel fibers BM = basilar membrane Nerve staining 880 ×*

outer spiral bundles (not visible in the picture) At this stage of degeneration, reduction in the nerve fiber population of the lamina spiralis ossea may also be seen

In specimens with most advanced *neural damage the outer spiral bundles* had completely disappeared some fibers of the inner spiral bundle and of the spiral tunnel bundle being left *No noteworthy differences were found at late stages between* different coils of the cochlea Degeneration in the animals with longest survival times was *studied after complete loss of sensory cells, however the progress of neural degeneration* was almost parallel in all parts of the cochlea

*Descriptions of the innervation of the organ of Corti* have usually distinguished two main types of nerve fibers namely, short radial orthoneurons and long spironeurons with a *spiral course (Retzius 1884 Lorente de No, 1937 Fernandez, 1951)* The majority of the orthoneurons are supposed to end at the inner hair cells branching there *and innervating a few neighboring cells* Only a few of them would pass the tunnel of Corti to terminate on outer hair cells The second type of nerve fibers the spironeurons bend at right angles and run considerable distances in the inner and outer spiral bundles The outer hair cells would be innervated preponderantly by these spiral fibers, whereas the inner hair cells would be innervated predominantly by the radial orthoneurons (*Fernandez,* 1951) The spiral fibers of the inner spiral bundle are believed to represent terminal fibers of the efferent Rasmussen olivo-cochlear bundle (*Fernandez* 1951) In surface specimens stained with the present technique we have often found long spiral fibers running in the lower part of the outer spiral bundles and se[illegible]ng branches to outer hair cells usually to cells belonging to the same row over [illegible]derable distance These fibers have been found in both normal and damaged [illegible] but are not discernible in all specimens They are further discussed by *En*[illegible] *Ules* and *Andersson*




FIG 34 — *Guinea pig Su 726 R neomycin 200 mg/kg body weight for 8 consecutive days sacrificed 35 days after last injection Surface preparat on 31/4 coils from base of cochlea Severe degeneration of inner spiral bundle (ISB) Only remnants of the inner nerve endings are seen and the course of individual long spironeurons can be followed Small swell ngs in the spironeurons conta ning mitochondria can be seen In this specimen the spiral tunnel bundle (STB) is relatively well preserved Rarefact on among the nerve fibers in the lamina spiralis ossea (LF) in the upper part of the picture Nerve staining 990 ×*

The course of the spironeurons can be more easily studied in specimens in which advanced degeneration of neural elements has taken place This holds true for both outer spiral bundles (Fig 31) and inner spiral bundle (Fig 34) and evidently depends upon the fa t that many of the nerve fibers and endings have undergone degener ation thus nproving visualization of the remaining fibers by el minating most of the interf ence In addition to the remnants of nerve endings black stained accumu lations of mitochondria are seen in the individual fibers (Fig 34) In regions where branching occurs especially clusters of mitochondria seem always to be present

The last remaining fibers of the spiral bundles outer inner and tunnel usually run sp rally for considerable distances It seems that normal specimens these bun dles must contain large numbers of fibers which l only a short course within the bund es These the orthoneurons apparently nerate earlier leaving only the spironeurons in specimens with advanced dam Thus at a certain late stage of degeneration only spironeurons remain with the organ of Corti the radial ortho neurons having completely disappeared

# VI DISCUSSION

Histologic changes in the cochlea resulting from neomycin and kanamycin have been reported in the literature by many authors. The present consensus seems to be that the sensory cells of the organ of Corti are the elements primarily affected by these drugs. The outer hair cells have been found to be more vulnerable than inner hair cells in neomycin treated animals (*Ruedi* et al 1953, *Hawkins* and *Lurie*, 1953, *Riskaer* et al 1956, *Olivieri* and *Rossi*, 1958), however, one study (*Lindsay* et al 1960) has reported the reverse to be true in human patients. The most severe and extensive damage has been found in the basal and second coils by all of these authors. In general, findings of the same type, i.e. greater vulnerability of outer hair cells and most pronounced damage in the basal part, have been reported after administration of kanamycin both in experimental animals (*Hawkins*, 1959, *Mesolella* and *Costa*, 1960, *Ward* and *Fernandez*, 1961, *Catalano* et al 1961, *Darrouzet* and *De Lama Sobrinho*, 1962, *Ardouin* et al., 1963) and in humans (*Benitez* et al 1962, *Jorgensen* and *Schmidt*, 1962). Earlier reports on cochlear changes caused by framycetin are not available.

These authors have furnished valuable information on the general features of cochlear damage from ototoxic drugs, e.g. that the basal part of the cochlea is more severely damaged and there appears to be a clear-cut difference in vulnerability between outer and inner hair cells, but no information on the exact pattern of cellular loss can be found in these papers. After administration of kanamycin *Beck* and *Krahl* (1962) found that in the guinea pig the two inner rows of outer hair cells undergo degeneration earlier than the outermost row. *Hawkins* and *Engstrom* (1964), using the same techniques as in the present study, showed that all outer hair cells in the basal coil and those in the first row of the third coil were less resistant to the drug than are other outer hair cells. The present findings support these results and confirm them as a partial picture of the degeneration pattern of cochlear sensory cells. In the present material the administration of neomycin, kanamycin and framycetin caused a closely similar pattern of sensory cell degeneration. The outer hair cells of the basal coil were damaged first. Degeneration then progressed upwards in the cochlea, affecting the outer hair cells of the two inner rows in the second coil and of the innermost row in the two apical coils, but at this stage leaving undamaged the two outer rows in the upper coils of the cochlea. The degeneration of inner hair cells also followed a definite pattern which, in one sense, is opposite to that of the outer hair cells, *beginning at the apex and progressing toward the base*. This indicates that not all inner hair cells are less vulnerable than all outer hair cells, as generally believed, although their degeneration begins later. It should be remembered there is a stage at which all inner hair cells of the apical coil are severely damaged while the majority of outer hair cells have still been spared. Slight deviation from this general pattern was encountered in a few animals treated with kanamycin. These showed a complete loss of sensory cells in the second coil at a relatively early stage of degeneration.

One of the major aims of the present study was to search for differences in the normal morphology of the cochlea which might correlate with the degeneration pattern described above Such a correlation was found to exist between degeneration pattern and innervation of the sensory cells as revealed by use of the modified zinc iod de osm um tetroxide stain of *Maillet* (1963) The existence and distribution of two types of nerve endings in the cochlea were known from electron microscopic studies (*Engstrom* 1958 *Smith* and *Sjostrand* 1961) but application of the *Maillet* stain to the cochlea made it possible to survey the whole neural pattern of the organ of Corti with light microscopy It was found that the degeneration pattern of outer hair cells has an almost exact correlation with the distribution of large richly granulated nerve endings In fact cochleograms from an appropriate stage of outer hair cell loss (Figures 24 and 25) could be used equally well to portray distribution of large nerve endings under outer hair cells In fact an almost similar schematic picture has been published by *Smith* and *Sjostrand* (1961) to show the distribution of large nerve endings It seems however that the outer hair cells are not actually divisible into two sharply differentiated types with respect to size of nerve endings Rather, there are two gradients one linear (or spiral) the other radial with respect to the cochlea as a whole such that nerve ending size diminishes from the base toward the apex and from the first or innermost toward the third or outermost row of outer hair cells The pattern of degeneration of outer hair cells shows the same gradients expressed as it were in terms of differential vulnerability For example although all of the outer hair cells of the basal coil show a relatively high vulnerability those of the first row are slightly though none the less definitely, more vulnerable than those of the second and third rows (Fig 19) It would be generally correct to say that the degree of vulnerability of outer hair cells is directly proportional to the size of nerve endings

Possible explanations of the differential vulnerability of outer hair cells in the different rows and coils of the cochlea have been discussed very little in the literature It has been suggested by *Beck* and *Krahl* (1962) that the slower blood flow in the basal part of the cochlea would result in locally higher concentration of the circulating drugs and thus the cells there might be damaged earlier However the clear-cut, systematic difference between cells adjacent to each other but belonging to different rows can scarcely be explained in this way It is the author s opinion that the correlation between degree of vulnerability and differential innervation of individual outer hair cells is so close that a more tenable explanation might be found in a hypothesis such as the following The richer the supply by large nerve endings (of a given hair cell) the more easily may its metabolism be disturbed by ototoxic drugs Support for a hypothesis of this kind may be found in the observation that several antibiot cs of the Streptomyces group including neomycin and kanamycin have an inhibitory effect on conduction across the myoneural junction and on synaptic transmission in autonomic ganglia (*Pittinger* and *Long* 1958 *Lullmann* and *Reuter*, 1960 *Brazil* et al 1961 *Osterloh* 1961 *Rean* 1963) This suggests that these drugs may have selective affinity for certain synaptic regions If the hair cell to nerve ending interface of the cochlea were one of these regions a correlation between hair cell vulnerability and synaptic area might be a reasonable possibility The histologic

# VI DISCUSSION

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methodology of this study of course does not provide evidence on the mode of attack of the drug on the sensory cells

A clear cut difference in vulnerability was also found between the inner hair cells at the apex of the cochlea and those at the base. Analogically, this might be based on a similar difference in innervation between inner hair cells in different parts of the cochlea. Such differences have not been reported as yet but studies on this subject are being carried on by *Engström* and his coworkers.

Morphological details of the neural damage within the organ of Corti as studied with light microscopy have not been previously reported. This omission is presumably due to technical difficulties in staining of the nerve fibers and nerve endings. In the present material the pattern of neural degeneration was found to follow very closely the pattern of cellular loss. Thus the degenerative changes first appeared in large endings of the outer hair cells and later in the nerve endings of the inner hair cells. Neural damage was never found without degeneration of corresponding hair cells.

A very characteristic morphological feature of neural degeneration is the clumping of disintegrating neural material into large irregular accumulations attached to the remaining nerve fibers (Figs. 51 and 52). These fibers have a spiral course in both inner and outer spiral bundles whereas the short radial orthoneurons at a late stage of degeneration have largely disappeared. A difference in vulnerability to ototoxic drugs between the two types of nerve fibers, spironeurons and orthoneurons, cannot be proved on the basis of these experiments; however it has been shown clearly that the course of individual fibers can be more easily studied in specimens in an advanced stage of degeneration. Thus observations on pathologic material can contribute materially to mapping of the normal neural pattern.

The spiral ganglia of the cochleas were not systematically examined, however in some sections made from cochleas with advanced damage in the organ of Corti no clearly identifiable changes were found in the spiral ganglion giving the author the impression that damage to the spiral ganglion is not necessarily associated with the early stages of neural degeneration within the organ of Corti. Further studies on this subject are needed.

*Engström*'s surface preparation technique (*Engström* et al. 1966) has proven to be a method eminently well chosen for evaluation of pathologic changes in sensory cells in this type of study where the primary aim is to define the overall pattern of damage. Many details of individual structures can also be discerned in surface specimens; however the method for this purpose supplements rather than replaces conventional serial sections of the organ of Corti. The use of the surface preparations as a basic method complemented by more detailed study of sections by light and electron microscopy provides a many-sided view of structural changes in cochlear degeneration which is far more comprehensive than any of the methods used singly could give. A not inconsiderable virtue of the surface preparation method is that it does not require expensive special equipment but only good phase contrast and dissecting microscopes supplemented by the [illegible] ordinary photomicrographic equipment.

Adequate [illegible] registering of cochlear cellular damage is an essential requirement of this [illegible] investigation which makes it possible to compare directly the results on ma[illegible] is subjected to different experimental conditions. The cochleo

gram technique, devised by Engstrom, fulfills the requirement for direct comparability, and is excellent as a means of rapidly surveying damage over wide areas Certain difficulties which are inherent and unavoidable in all methods of graphic reconstruction of the cochlea (*Guild*, 1921, *Schuknecht*, 1953), are circumvented by the cochleogram method an example being the easy identification of individual cells normal or damaged each with its proper row of hair cells Given a certain amount of practice reasonable accuracy can be achieved in registering the findings as seen under the microscope The method is undergoing further standardization to develop its full usefulness, to make it as simple in use as possible, and to minimize the chance of error in its application

The use of the pinna reflex threshold, stimulated by a Galton whistle, is admittedly a very rough hearing test It was used in this study, not in the expectation of obtaining reliable, correlative data on functional hearing, but because experience showed it to have a certain practical use as an indicator of when to sacrifice the animals with reasonable assurance that the pathologic process had advanced to a histologically significant degree Obviously, if one were to set out to correlate histologic changes in the cochlea to changes in hearing function, as must ultimately be done, some other, more accurate method of measuring hearing thresholds must be used

The daily drug dosages used in these experiments were very large as compared to clinical dosages used on human patients As measured in terms of ratio of drug dose to body weight, the animals received 15–30 times the normal human dosage Despite this, the animals ordinarily did not display any signs of severe general toxicity, except for transient slight weight loss early in the series of injections Hence, the systematic damage which occurred in the cochleas cannot reasonably be attributed to general toxicity of the drugs, but rather must be accounted for on the basis of a more specific ototoxicity Nevertheless as a further control on the hypothetical factor of general toxicity, further studies have been undertaken in which equivalent amounts of the drug are given by smaller doses over a longer period of time

Previous histologic work with other antibiotics of the Streptomyces group (e g *Ruedi* et al , 1951, *Hawkins* and *Lurie*, 1952, 1953, *Riskaer* et al , 1956) suggest that a very similar pattern of damage will be found if the surface preparation technique is applied to the study of cochlear damage from these drugs Only further experiments will determine whether the pattern described is specific to the antibiotics of the Streptomyces group or instead expresses a general tendency in the degeneration of cochlear sensory cells no matter what noxious agent is responsible

# VII SUMMARY

In the review of the literature the ototoxic properties of the three antibiotics concerned in this study, neomycin kanamycin and framycetin were discussed Special attention was given to previous experimental histologic studies with these drugs

The purpose of the present study was to search for a systematic pattern in the distribution of cochlear sensory cell damage from the three ototoxic drugs and if such a pattern can be defined, to determine to what degree it may correlate with structural differences present in the normal cochlea A study of degeneration of neural elements within the organ of Corti was made paralleling that of sensory cell damage in order to correlate pathologic changes in the two structures with each other

The experimental material consisted of 58 guinea pigs injected with systematically varied doses of the three drugs and allowed to survive for different periods after the treatment Most of the cochleas were prepared by the surface preparation technique of *Engstrom* (*Engstrom* et al, 1964) Part of the surface specimens were studied under the phase contrast microscope after osmium fixation for evaluation of the cellular damage part of them stained with a new nerve impregnation technique modified from the original method of *Maillet* (1963) From the surface specimens the loss of sensory cells was graphically registered using *Engstrom* s cochleogram method A few of the cochleas were embedded in acrylate or epoxy resin and studied as sections by light or electron microscopy

The morphology in the degeneration of individual sensory cells was briefly described and discussed The pattern of hair cell damage in the cochlea as a whole was presented by series of micrographs and cochleograms showing stages of degeneration which ranged from slight scattered loss of cells to an almost complete destruction The pattern was found to follow the same general principles with each of the three drugs The outer hair cells of the basal coil were affected first the damage then progressed apicalward in the cochlea affecting the two inner rows of the second coil and the innermost row of the two upper coils The degeneration of inner hair cells also followed a clear-cut pattern beginning at the apex and progressing towards the base

The pattern of neural damage was found to follow very closely the damage to the sensory cells Degeneration of nerve endings or nerve fibers appears very soon after the loss of corresponding hair cells and neural damage was never found without loss of sensory cells Clumping of the disintegrating neural material into irregular accumulations attached to the remaining nerve fibers was found characteristic of late stages of degeneration In specimens with advanced degeneration the short radial nerve fibers or orthoneurons had almost completely disappeared so that the course of the long spironeurons in the inner and outer spiral bundles could be studied much more easily than in normal specimens

In the discussion of the results the main emphasis was laid on the close relation of the pattern of outer hair cell loss to the distribution of the large richly granulated nerve endings adjacent to the outer hair cells. The vulnerability of an individual outer hair cell was found directly correlated to the supply of large nerve endings, the cells with the richest innervation with large endings being the most vulnerable, and the cells innervated with small endings only, the most resistant. Between these two extremes a gradation in vulnerability was found which corresponded with gradation of innervation. Comparable structural differences between the most vulnerable inner hair cells (those at the apex) and the most resistant (those at the base) are not yet apparent.

The final discussion had to do with whether the degeneration pattern observed is specific to the Streptomyces group of antibiotics or may rather express a general tendency in cochlear hair cell degeneration, regardless of the noxious agent. The available evidence does not allow a decision, and comparable data on other noxious agents (e.g. noise exposure) will be needed in order to solve this question.

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# A STUDY OF THE ACOUSTIC REFLEX IN INFANTRYMEN

BY

MICHAEL H. L. HECKER
AND KARL D. KRYTER

ACTA OTO-LARYNGOLOGICA

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SUPPLEMENTUM 207

# A STUDY OF THE ACOUSTIC REFLEX IN INFANTRYMEN

MICHAEL H L. HECKER and KARL D KRYTER

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## ABSTRACT

The degree of reflex response to monaurally presented white noise (100 dB SPL) was measured and graphically recorded at the contralateral ear for 40 career infantrymen with an acoustic bridge These soldiers had been exposed to controlled amounts of weapon noise and pre and post exposure audiograms were obtained The results show that subjects with a high pre exposure HI are less susceptible to TTS than subjects with normal hearing The results further indicate that a strong acoustic reflex is associated with high rather than low pre exposure HI for subjects having no appreciable conductive hearing impairment Subjects with suspected middle ear disorders exhibited no reflex response to the same stimulus

## INTRODUCTION

Although the nature of the acoustic reflex is by no means fully understood the many experiments that have been carried out during the past decade suggest that the reflex phenomenon may be practically valuable in at least three respects First there is evidence that the action of the middle ear muscles attenuates certain kinds of intense acoustic signals and thus protects the cochlea from acoustic trauma Both animal [4 13] and human ears [3 16] have been studied in this respect Second the reflex action can provide useful information in the clinical diagnosis of hearing disorders A difference of less than 70 90 dB between the threshold of hearing and the reflex threshold (lowest intensity of a tone that will elicit the reflex) indicates the presence of recruitment [2, 11 15] And finally recent research suggests that the acoustic reflex should be considered in explaining the results of various psychoacoustic experiments particularly those involving masking and loudness scaling [10]

Because it has been demonstrated that the reflex serves to protect the inner ear when subjects are exposed to a rapid succession of intense acoustic transients (such as gunfire), the hypothesis has been formulated that in a population of subjects who are constantly exposed to such transients those individuals with a strong reflex will tend to have less noise induced hearing loss than individuals with a weaker reflex In order to investigate possible

general relations between a measure of reflex activity, auditory fatigue, and permanent hearing loss, an experiment was performed in which 10 U.S. Army infantrymen participated as subjects.

These soldiers were made available following a government program for the evaluation of new weapons [8]. In this program the men had been exposed to controlled amounts of weapon noise, and audiograms had been obtained prior to and just after exposure. The earlier program thus provided pre-exposure hearing level (HL) and temporary threshold shift (TTS) data for both ears of the test subjects.

The acoustic reflex of each infantryman was measured by means of an acoustic bridge that was developed by Zwislocki [17] and is manufactured by the Grason-Stadler Company (Model E8872A). With this instrument the acoustic impedance of the eardrum can be measured at various frequencies. When the acoustic reflex is elicited by means of a noise presented to the opposite ear, the impedance measurement in the near ear will change from its previous value. The degree of this change is an indication of the amount of middle-ear muscle activity. It is possible to elicit the reflex in one ear and to measure it in the other ear (contralateral measurement) because the reflex is normally a bilateral response to a unilateral stimulus.

The acoustic impedance method of measuring the reflex also permits the *taking of measurements in the same ear where the reflex is elicited* (homolateral measurement), but this technique increases the complexity of the instrumentation and was not used in the present study.[1] Another method of measuring the acoustic reflex employs an instrument which is sensitive to the dynamic displacement of the eardrum (tympanomanometer). This method is restricted to contralateral measurements because the ear canal into which the differential pressure probe is inserted must be tightly sealed.

## PROCEDURE

Both ears of each test subject were examined by an otologist before acoustic impedance measurements were taken. This was done so that possible infections or perforations of the eardrums, which might interfere with or prohibit the test procedure, would be detected. Also, any otological history, or history of childhood diseases which may have affected the

[1] Møller has demonstrated that the degree of reflex activity is somewhat greater for homolateral than for contralateral measurement of the reflex [12]. However, techniques employing contralateral measurement are valid and more practical whenever normal subjects are being tested. (When a subject with a unilateral conductive hearing loss or with a unilateral pathology in the neural pathways of the reflex are is being tested, the response measurements in a given ear depend greatly upon whether the reflex-arousal sound is presented to that ear or to the opposite ear.)

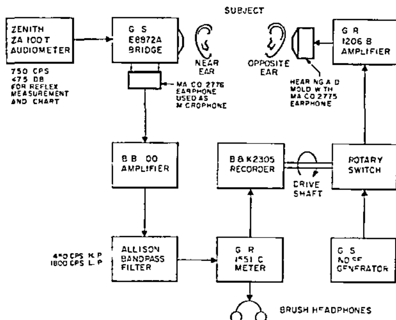



Fig 1 Schematic diagram of instrumentation used to measure and chart muscle reflex at 750 cps

middle ears, was recorded wherever applicable Then the ear canal into which the acoustic bridge was to be fitted was thoroughly cleaned

The actual measurement session for each subject consisted of two parts First, the acoustic impedance of the eardrum of the near ear[1] was measured at 250, 500, and 1000 cps in the conventional manner [17] The test tones were generated by a Zenith (Model ZA100T) portable audiometer and monitored through the stethoscope supplied with the bridge For the second part, the stethoscope was replaced by a microphone whose output was amplified and band pass filtered so that a test tone could be graphically recorded The impedance was measured at 750 cps and the test tone was now monitored through a pair of headphones Then a steady reflex-arousal sound (100 dB SPL of white noise) was presented to the opposite ear through an earphone fitted to a hearing-aid mold, and the impedance measurement was repeated Finally, the bridge was adjusted again to the values obtained without the reflex and the test tone was graphically recorded while a particular pattern of reflex-arousal noise was being generated automatically

A schematic diagram of the instrumentation used to measure and record the impedance changes produced by the acoustic reflex is shown in Fig 1. The 750 cps test tone generated by the audiometer and supplied to the bridge was not permitted to exceed 75 dB (audiometer attenuator setting)

[1] The near ear is defined as the left ear for right handed firers (who place the butt of their weapon against their right shoulder) and as the right ear for left handed firers

so that it could not elicit the reflex. The idea of replacing the stethoscope with a microphone is not new: both Zwislocki and Tilly [9] have undertaken exploratory studies with an electrical output from this particular bridge.[1] From this figure it can be seen how the noise pattern is automatically generated by means of a special rotary switch that is coupled mechanically to the Bruel + Kjaer (Type 2305) level recorder.

The intensities of the 750 cps test tone and the reflex arousal noise and all amplifier gain settings were kept constant throughout the experiment. Some difficulty was experienced during the recording of the output from the bridge. A few soldiers could not be properly fitted with any of the available hearing aid molds, so that some reflex arousal noise from the opposite ear radiated to the microphone at the near ear. Noise picked up in the 450–1800 cps passband was consequently amplified and more or less obscured the recorded bridge output. In these cases, in order to determine how much of the recorded signal is contributed by the test tone and how much by the noise, a portion of the recording was made with the audiometer disconnected (no bridge excitation).

## RESULTS

### Acoustic Reflex

Impedance measurements have been obtained for the near ear of each subject at 250, 500, 750, and 1000 cps. Since impedance is a complex quantity, a given measurement produces two numbers which express the compliance (related to springiness) and the resistance (related to damping) of the eardrum. On the acoustic bridge the compliance scale is calibrated directly in $cm^3$ of equivalent air volume, but the resistance scale is in arbitrary units which may be converted to acoustic ohms by means of a calibration chart. In addition to quiescent impedance measurements, the reductions in compliance and resistance at 750 cps produced by the sustained contraction of the middle ear muscles were obtained for each subject. The reductions in compliance and resistance may be expressed respectively in terms of a compliance ratio (C.R.) and a resistance ratio (R.R.). Ratios of unity would indicate that no change was recorded and that the acoustic reflex was not present.

[1] In the present study a single frequency (750 cps) is monitored with the electrical system. A variety of problems are encountered when an attempt is made to monitor several frequencies electrically. The dimensions of the coupling between the acoustic bridge and the microphone are such that the electrical frequency response of the system varies over a 40 dB range from 125 to 1[illegible]00 cps. It is extremely difficult [illegible] possible to balance the bridge at frequencies [illegible] to 500 cps.

TABLE 1 *Criteria for middle-ear normalcy Compliance expressed in cm³ of equivalent air volume, resistance in acoustic ohms Values outside of these ranges suggest possible middle ear pathology*

| | Frequency (cps) | | | |
|---|---|---|---|---|
| Criteria | 250 | 500 | 750 | 1000 |
| Compliance range | 0.35 ↓ 2.3 | 0.35 ↓ 3.5 | 0.50 ↓ 5.0 | 0.60 ↓ >5.0 |
| Resistance range | 60 ↓ 1500 | 120 ↓ 850 | 120 ↓ 1200 | 120 ↓ 600 |

Zwislocki has developed tentative criteria for middle-ear normalcy [17, 18] which are given in Table 1 If several compliance values have been obtained which exceed the lower limits of the compliance range [18], and the associated resistance values exceed the upper limits of the resistance range [17], otosclerosis should be suspected Similarly, if several compliance values exceed the upper limits of the compliance range [17] and the resistance values exceed the lower limits of the resistance range [17], ossicular discontinuity (rare) should be suspected Examination of the impedance measurements suggested that six soldiers had a possible middle-ear pathology

The remaining 34 soldiers with apparently normal middle ears (near ears) were then classified into three subject categories according to the degree of reflex activity that could be measured in their near ears The

TABLE 2 *Criteria for subject categories The compliance ratio (C R) and resistance ratio (R R) of each subject in a given category fall within the ranges indicated*

| Category | No of subjects | Criteria |
|---|---|---|
| I Pronounced reflex | 7 | C.R ≤ 0.55 or 0.55 < C.R ≤ 0.65 R.R < 0.80 |
| II *Some reflex* | 10 | 0.55 < C.R ≤ 0.65 *R.R > 0.80* or 0.65 < C.R ≤ 0.80 |
| III No reflex | 17 | 0.80 < C.R ≤ 1.00 |
| IV Abnormal | 6 | Suspected pathology in near middle ear |

so that it could not elicit the reflex. The idea of replacing the stethoscope with a microphone is not new; both Zwislocki and Lilly [9] have undertaken exploratory studies with an electrical output from this particular bridge.[1] From this figure it can be seen how the noise pattern is automatically generated by means of a special rotary switch that is coupled mechanically to the Bruel + Kjaer (Type 2305) level recorder.

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having a duration of less than perhaps 100 msec But adequate protection appears to be provided for the last two of the three 300 msec bursts of noise After the second long (1 65 sec) burst of noise the audiometer was disconnected, and the remainder of the recording for this subject shows the amount of noise leakage from the opposite ear as picked up by the microphone attached to the bridge

A resultant contraction time of perhaps 100 msec has been recorded for the subject representing Category II, but his acoustic reflex is seen to be weaker than that of the subject from Category I The subject representing Category III displays a similar resultant contraction time (100 msec) on those occasions when a very weak reflex could be elicited The three recordings shown in this figure were obtained from subjects who could be provided with tight fitting hearing aid molds for their opposite ears, and consequently the reflex-arousal noise did not obscure the traces of the 750 cps test tone for these subjects

## Audiometric Data

The audiometric data for the near and opposite ears of the 40 soldiers are presented in Figs 3 and 4 (The near ear is the left ear and the opposite ear the right ear for right handed firers ) For each ear of each subject the temporary threshold shifts measured at 3 and 4 kcps were converted to values that would have been obtained 2 minutes after exposure to the weapon noise (TTS ) and then averaged This average $TTS_2$ is plotted as a function of the pre exposure hearing level (HL), which was also averaged at 3 and 4 kcps In these figures the entries are coded according to subject category in order to illustrate the wide spread of the results for subjects in a given category

Before the audiometric data were processed further, a convention was established and imposed with respect to small and negative values of TTS, All values between plus 4 and minus 4 dB were arbitrarily called *zero* Values below minus 4 dB were also called zero, it was suspected that for *these subjects head noises (tinnitus), produced by the exposure, had inter*fered with the audiometry All values of $TTS_2$ above plus 4 dB were left unaltered For reference purposes this convention is noted in the right hand margin of Figs 3 and 4

It is unfortunate for this experiment that all subjects were not exposed to the same weapon noise Two different types of weapons and various firing schedules were assigned to these soldiers when they participated in the earlier weapon-evaluation program [8] For this reason the TTS values obtained for different subjects cannot be directly compared in an attempt to show differences in susceptibility to auditory fatigue

To partially overcome this shortcoming, the $TTS_2$ averages for the four subject categories were corrected for equivalent exposure to all categories by means of the following procedure First, the $TTS_2$ values obtained in

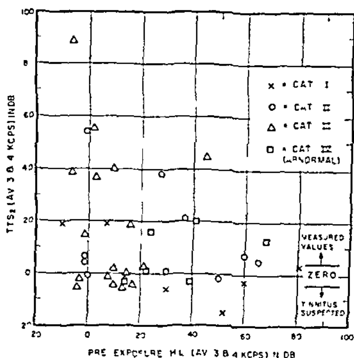



Fig. 3 $TTS_2$ as a function of pre-exposure HL for 40 near ears. Data obtained from pre- and post-exposure audiograms.

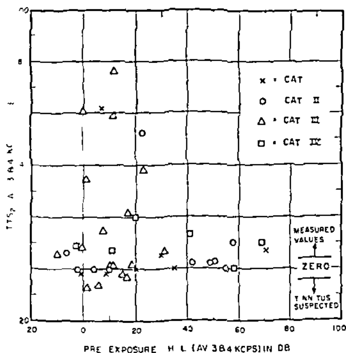



Fig. 4 $TTS_2$ as a function of pre-exposure HL for 40 opposite ears. Data obtained from pre- and post-exposure audiograms.

**TABLE 3** *Predicted $TTS_2$ for each subject category and for all subjects, based on audiometric data (averaged at 3 and 4 Kcps) obtained from a larger population of soldiers using the same types of weapons and firing schedules as the test subjects [8] $TTS_2$ correction factors for under or over-exposure relative to the average for all categories are also indicated*

| | Subject category | | | | |
|---|---|---|---|---|---|
| | I | II | III | IV | All |
| Predicted $TTS_2$ in dB | 16 | 12 | 18 | 16 | 16 |
| Correction, dB | 0 | +4 | −2 | 0 | 0 |

the weapon evaluation program for each group of soldiers using a particular weapon and firing schedule were averaged and adjusted to take into account group differences in pre exposure HL These data represent, then, average TTS values that can be predicted from exposure to various combinations of weapon and firing schedule Second, the appropriate predicted $TTS_2$ value was recorded for each of the 40 test subjects and averages were obtained for the four subject categories These predicted averages and $TTS_2$ correction factors derived from them are shown in Table 3

With reference to Table 3, one would expect—other things being equal, such as pre exposure HL, middle-ear impedance and acoustic reflex—that the $TTS_2$ average for Category II will be about 4 dB lower than the $TTS_2$ average for Category I, because the soldiers in Category II happen to have been exposed to firing conditions which produce, on the average, less $TTS_2$ than the firing conditions to which the soldiers in Category I were exposed

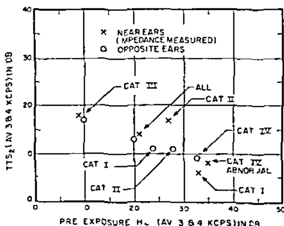



Fig 5 $TTS_2$ as a function of pre exposure HL, averaged for ears in each subject category and for all categories (40 ears) Corrected for equal exposure

Figure 5 shows the averaged $TTS_2$ and pre-exposure HL for each subject category, including the $TTS_2$ correction factors listed in Table 3. Data for near ears and opposite ears are shown separately. Five tentative observations can be made from this figure which are presented below as statements, the first four of these statements will be discussed in the next section of this report.

1. Ears having a high pre-exposure HL tend to be less susceptible to TTS than ears having a lower pre-exposure HL. The relation between $TTS_2$ and pre-exposure HL is nearly linear and has *an approximate slope of −8 dB* $TTS_2$/20 dB pre-exposure HL.

2. Subjects (Category III) whose near ears give normal impedance measurements (no appreciable conductive hearing impairment) but who possess a weak acoustic reflex (as measured at the near ear and elicited at the opposite ear) tend to have a low pre-exposure HL and a high $TTS_2$. Conversely, subjects (Categories I and II) with a stronger reflex tend to have a high pre-exposure HL and a low $TTS_2$.

3. Because the test subjects fired their weapons only once every 5 seconds (presumably the middle-ear muscles were relaxed at the onset of each impulse), the present experiment did not bear on the question of whether the acoustic reflex tends to protect against high TTS. However, the results suggest that the reflex did not adequately protect the hearing of these soldiers during their Army careers.

4. Subjects (Category IV) whose near ears are categorized as abnormal on the basis of impedance measurements (conductive hearing impairment suspected) tend to have the highest pre-exposure HL in both ears and tend to possess no acoustic reflex.

5. Although the data for near and opposite ears differ somewhat for Categories I and II, the pre-exposure HL and $TTS_2$ averages for the two ears are highly correlated.

## DISCUSSION

The relation between $TTS_2$ and pre-exposure HL is the subject of the first observation. This relation, with approximately the same slope, is also encountered in studies of TTS induced by steady state industrial noise and has been reported previously [5]. Also, the same relation and slope can be demonstrated for this study by grouping the 40 subjects according to pre-exposure HL (instead of reflex category). This was done, but the results are repetitious and therefore not included in this report.

The subject matter of the second observation is more controversial. Those subjects who have a high pre-exposure HL but no appreciable conductive hearing impairment are suffering from the noise-induced (sensory-neural) loss that is typically associated with career infantrymen. However, the lowest intensity of the reflex-arousal sound which will first elicit the

TABLE 4 *Average acoustic reflex of near and opposite ears expressed in terms of compliance ratio (C R ) and resistance ratio (R R ), for four ranges of pre exposure HL (averaged at 3 and 4 Kcps)*

| Pre X HL range (dB) | Near ears | | | Opposite ears | | |
|---|---|---|---|---|---|---|
| | No ears | C R | R R | No ears | C R | R R |
| ≤ 0 | 10 | 0 84 | 0 84 | 9 | 0 87 | 0 83 |
| 1–19 | 13 | 0 88 | 0 90 | 15 | 0 85 | 0 93 |
| 20–39 | 8 | 0 79 | 0 92 | 7 | 0 74 | 0 82 |
| ≥ 40 | 9 | 0 75 | 0 81 | 9 | 0 78 | 0 84 |

acoustic reflex in these subjects (reflex threshold) is known to be, on the average, *the same as in subjects with normal hearing (70–90 SPL) [11]* This fact is attributed to the complete recruitment of loudness at these levels that is characteristic of most sensory neural losses [1, 6] It may be assumed, therefore, that the reflex-arousal noise was perceived equally loud by nearly all subjects in Categories I, II, and III

In order to examine whether the observed association of high pre exposure HL with strong acoustic reflex might not be an artifact introduced, perhaps, by the particular criteria used for establishing the subject categories, all 40 subjects were grouped according to their pre exposure HL The average acoustic reflex was calculated for four ranges of pre-exposure HL, separately for both ears The results, which are shown in Table 4, are in agreement with and thus serve to confirm the observation

If a significant number of the subjects with a high pre exposure HL had over-recruitment of loudness, then the 100 dB SPL reflex-arousal noise would have been perceived louder by these subjects than by subjects with a lower pre exposure HL (and less recruitment) This increased loudness, in turn, would have elicited a stronger reflex But it appears that over recruitment is not encountered in acoustic trauma cases, although it is common with Meniere's disease [6, 11] It should be noted, however, that the degree of recruitment is determined with the aid of specific clinical tests (developed by Fowler and by Reger) which do not, and were not intended to, explore all aspects of the intensity loudness relation

Even if it can be assumed that both normal hearing subjects and subjects with a sensory neural loss have the same intensity loudness relation above the reflex threshold, the relation between loudness and degree of reflex response may still be quite different for the two populations Well above the intensity corresponding to reflex threshold, then, it might be possible to elicit a stronger reflex from subjects with large sensory-neural losses than from subjects with smaller losses Perhaps the stronger acoustic reflex is able to reduce pain which may be produced by the stimulus in subjects with large losses Not enough is known at present about the relation between loudness and degree of reflex response for different hearing disorders, and the need for more research is indicated

There are reasons, however, to question the assumption that the intensity-loudness relation above reflex threshold is indeed the same for all degrees of sensory-neural loss. In the clinical recruitment tests a 4 Kcps tone is usually employed, and it is known that different recruitment charts can be obtained when loudness balances are made at different frequencies or with wide-band or narrow-band noise. Therefore, the absence of clinical over-recruitment (at 4 Kcps) in subjects with noise-induced hearing loss does not necessarily mean that these subjects perceive a reflex-arousal sound which is not a 4 Kcps tone with the same loudness as subjects having normal hearing.

Also, the general failure of clinical tests to demonstrate over-recruitment can perhaps be explained as follows. Noise-induced hearing loss is usually bilateral and the monaural version of the recruitment test must therefore be employed. In the monaural test a high-frequency tone is matched in loudness to an alternately presented low-frequency reference tone for which the loss is smaller. Since a high-frequency tone is known to be effective in eliciting the acoustic reflex and the attenuation provided by the reflex is greater for low than for high frequencies, the loudness of the low-frequency reference tone is handicapped whenever this tone is switched on. (Presumably the reference tone is not presented long enough for the middle-ear muscles to relax.) Consequently, subjects with a strong reflex may actually have over-recruitment[1] but their recruitment charts will show the same intensity-loudness relation above the reflex threshold as charts obtained from normal-hearing subjects with a weaker reflex. Again, more research is indicated in this area.

Terkildsen reported a study which is somewhat related to the subject under discussion [14]. He compared measurements of the acoustic reflex obtained from 18 workers exposed for years to intense industrial noise (at 4 Kcps the average HL was 42 dB) with measurements obtained from 50 subjects having normal hearing. Both groups exhibited the same average reflex threshold (75 dB SPL) and the same average "resultant contraction time", but the average degree of reflex response was slightly less for the workers group. While this finding is at variance with the present results, the two studies differ in several important respects. In the first place, Terkildsen used different instrumentation. Also, he employed a 1 Kcps reflex-arousal tone rather than broad band noise. And finally, there was a considerable difference between the average ages of the workers group (48 years) and the group with normal hearing (28 years). The average age of the infantrymen who participated in the present study was about 24 years.

The third observation made in the previous section of this report deals with the protective function of the acoustic reflex. The data shown in

[1] Perhaps over recruitment can be demonstrated in these cases if the acoustic reflex is elicited by non acoustic means during the entire test.

Fig 5 do not demonstrate that the reflex tends to protect against high TTS, because of the effect of different pre-exposure HL If the TTS, averages shown for the subject categories are adjusted to correspond to some common pre-exposure HL, using the −8 dB TTS₄/20 dB pre-exposure HL slope, all values of TTS₄ will converge In this particular study the absence of protection is not surprising, however, because the firing schedules were purposely designed to exclude the acoustic reflex as an experimental variable (Each soldier pulled the trigger of his weapon once every 5 seconds for the duration of the exposure )

Fletcher and Riopelle have clearly demonstrated the protective function of the reflex for controlled exposures to weapon noise in terms of reduced TTS when the weapon is fired during contraction of the middle-ear muscles [3]. But the limitations of reflex protection against the type of weapon noise ordinarily encountered by infantrymen have also been reported [10, 16]

Because of the relatively long contraction time of the muscles, no attenuation is provided for acoustic impulses having a short rise time if they are separated by at least 1 second (present study) Some protection is provided for all but the first impulse if the impulses are spaced closer in time But since the reflex action attenuates low frequencies much better than high frequencies, and Fourier Analysis of these impulses reveals the presence of strong high frequency components, this protection is relatively inefficient If the impulses occur in rapid succession, as in the case of sustained machinegun fire, a gradual adaptation of the reflex takes place and the cochlea is again offered little protection

It is reasonable, therefore, to expect that the acoustic reflex may not be effective in preventing career infantrymen from developing a permanent hearing impairment The results of the present study certainly support this expectation They also suggest the possibility that the level of an individual s reflex response gradually increases as he continues to be exposed to noise and eventually acquires a noise-induced hearing loss

The fourth observation made earlier concerns those subjects who have a suspected conductive hearing impairment (Category IV) The proximity of the plots for the near and opposite ears of these subjects in Fig 5 suggests that the conductive impairment was mainly bilateral No appreciable acoustic reflex could be measured in the near ears because of the abnormally enhanced reflex threshold that is characteristic of cases with conductive impairment (absence of loudness recruitment) [1]

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S U P P L E M E N T U M 2'9

# A GUIDE TO NEURO-OTOLOGICAL DIAGNOSIS FOR THE PRACTICING OTOLARYNGOLOGIST

BY
SIDNEY N. BUSIS

ACTA OTO-LARYNGOLOGIC

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SUPPLEMENTUM 209

# A GUIDE TO NEURO-OTOLOGICAL DIAGNOSIS FOR THE PRACTICING OTOLARYNGOLOGIST

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UPPSALA 1965

Presented as a candidate's thesis
to the American Laryngological,
Rhinological, and Otological Society

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# INTRODUCTION

Seldom does a day pass that the practicing otolaryngologist is not presented a *symptom which raises the question in his mind, "Could this be central?"* The aim of this discussion is to reinforce his clinical skill so that he can answer this question with a greater degree of security. Hopefully, this presentation will aid the otolaryngologist in the further development of a *clinical sense which embraces the concept of thinking of the* neuro-otological patient within the framework of inter-related otological, neurological, and vascular elements. With a broader view of these interrelationships, the otolaryngologist is better equipped to examine a patient, who presents symptoms which may represent primary otological disease or primary neurological disease, and come to a reasonable conclusion. He should arrive at the diagnosis of a disease which the otolaryngologist can treat, or a disease that requires treatment by another physician, or have the confident knowledge that further studies must be performed.

## ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

(3, 4, 6 20, 39)

### BRAIN STEM

To aid in understanding the various problems in neuro-otology, it is helpful to have a gross appreciation of the brain stem (Fig 1) which is, in a sense, the keystone of neuro-otology With the cerebellum, the brain stem occupies the posterior cranial fossa and is separated from the middle cranial fossa by the tentorium cerebelli The brain stem is comprised of the mid brain the pons and the medulla oblongata The mid brain includes the cerebral peduncles anteriorly and the corpora quadrigemina (the superior and inferior colliculi) The pons is interposed between the mid brain and the medulla The medulla is continuous with the spinal cord Ventrally, it rests upon the basilar portion of the occipital bone The cerebellum is connected to the brain stem by the three cerebellar peduncles the superior or brachia conjunctiva the middle or brachia pontis and the inferior or restiform bodies The cavity between the cerebellum dorsally and the pons and medulla ventrally is the fourth ventricle It is continuous superiorly with the cerebral aqueduct connecting it to the third ventricle, and inferiorly with the central canal of the spinal cord It is also connected by three openings one medial and two lateral, with the subarachnoid space The brain stem contains the nuclei of all the cranial nerves except the first and second It contains the medial longitudinal fasciculus, a complicated bundle extending from the third nerve nucleus into the spinal cord It is near the mid line and beneath the aqueduct and the fourth ventricle It contains fibers from the vestibular nuclei, the eye muscle nuclei, and the basal ganglia The brain stem also transmits all fibers from the brain to the spinal cord and contains a multitude of connections between its various nuclei and other nuclei in the central nervous system The angle between the cerebellum and the brain stem is known as the cerebello-pontine angle (Fig 2) and contains the nerve trunks of the fifth, seventh eighth, ninth, tenth and eleventh cranial nerves

### THIRD FOURTH AND SIXTH CRANIAL NERVES

Although there are many connections between the labyrinth and the eye muscle nuclei, it is beyond the scope of this paper to explore this multitude of connections Suffice it to state that the eye muscle nuclei are in the

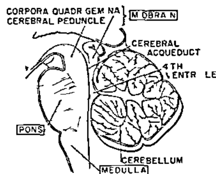



FIG 1 Brain stem (mid bra n pons and medulla) and cerebellum

brain stem near the midline connected to each other and associated balance receptors in the medial longitudinal fasciculus Lesions in the medial longitudinal fasciculus adjacent to it or in the eye muscle nuclei produce bizarre eye movements or paralyses such as dissociated eye movements unilateral nystagmus and disturbance in conjugate gaze (10 11) In summary the clinical finding of unilateral nystagmus means brain stem disease and the finding of conjugate deviation or dissociated eye movements means a central lesion which may be in the brain stem or in the supranuclear centers

### FIFTH CRANIAL NERVE (Trigeminal Fig 3)

The trigeminal nerve is the great sensory nerve to the face however it also is the motor supply to the muscles of mastication The cell bodies of the sensory portion are in the Gasserian ganglion on the cerebral surface of the apex of the petrous pyramid The sensory root enters the lateral pons in the upper part of the cerebello pontine angle The main sensory nucleus is in the upper portion of the pons however the sensory nucleus extends upwards toward the cerebrum and downward to the spinal cord The latter extension is known as the nucleus of the spinal tract of the




FIG 2 Cerebello-pontine angle

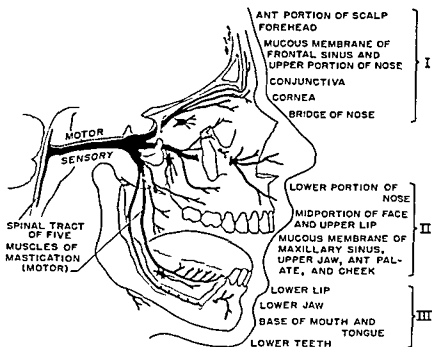



Fig 3 Fifth cranial nerve (Trigeminal)

trigeminal nerve (6) This is of clinical significance because the main nucleus is concerned with tactile sensation of the face whereas the nucleus of the spinal tract is concerned with pain The sensory supply to the cornea consists largely of pain fibers and, since the face is represented *in an inverted position in the nucleus of the spinal tract, the lower-most* portion of the nucleus which would be compressed by a cerebello-pontine angle tumor is responsible for corneal sensation and the corneal reflex This explains why cerebello-pontine angle tumors, once out of the internal auditory meatus and compressing the brain stem, ablate the corneal reflex before producing other sensory changes

Three sensory branches leave the Gasserian ganglion The first or ophthalmic division carries sensation from the forehead, anterior portion of the scalp, bridge of the nose, conjunctiva, cornea and mucous membrane of the frontal sinus and the upper portion of the nose The second or maxillary division carries sensation from the mid portion of the face and upper lip, the mucous membrane of the maxillary sinus and the lower portion of the nose and mucous membrane of the upper jaw and the anterior palate The third or mandibular division carries sensation from the skin of the lower portion of the face, that area which covers the mandible and lower lip, mucous membrane of the cheek, the lower jaw and the base of the mouth and tongue and the lower teeth

The motor nucleus is in the mid-pons medial to the sensory nucleus The motor portion leaves with the main sensory trunk and joins the

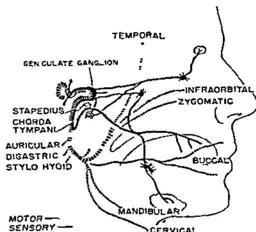



Fig 4 Seventh cranial nerve (Facial)

mandibular division to supply the muscles of mastication and the tensor tympani and tensor palati muscles

## Seventh Cranial Nerve (Facial, Fig 4)

The facial nerve consists of a larger motor portion and a smaller sensory root known as the nervus intermedius Both portions leave the brain stem in the cerebello pontine angle and enter the internal auditory canal in a common meningeal sheath with the eighth nerve The cell bodies of the sensory portion, nervus intermedius are found in the geniculate ganglion located on the seventh nerve just where it turns posterolaterally at the processus cochleoformis to traverse the middle ear The nervus intermedius also carries preganglionic secretory fibers to the lacrimal and submaxillary and sublingual salivary glands The sensory division carries taste sensation from the anterior two thirds of the tongue These fibers pass through the chorda tympani branch which joins the facial in its vertical canal inferior to the stapedial branch The sensory division also carries sensation from the concha which explains the pain and vesiculation of herpes zoster oticus (Ramsay Hunt Syndrome) It is believed that the sensory portion carries proprioceptive impulses from the facial muscles and deep pain sensation from the face (6)

The motor fibers supply the stapedius muscle with a branch which leaves the nerve in the upper part of its vertical mastoid portion and all the muscles of facial expression While there are several facial branches, it is best for clinical purposes to consider these in three groups, the forehead group the periocular muscles and the periorbital muscles Since the fibers to the forehead have bilateral supranuclear innervation, supranuclear lesions produce paralysis of the lower two thirds of the face while lesions of the nucleus or peripheral nerve produce paralysis of the entire ipsilateral side of the face

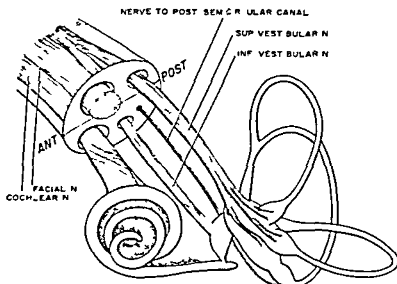



Fig 5 Seventh and eighth cranial nerves showing their position in the internal auditory canal passage through the foramina in the fundus of the canal and distribution to the labyrinth

## Eighth Cranial Nerve—Cochlear Division

The eighth nerve is considered in its two divisions (Fig 5) The cochlear division should be thought of in five components, the end organ cell bodies in the spiral ganglion, the nerve trunk, the cochlear nuclei, and the central connections from the nuclei to the cerebrum with way stations in the superior olivary nucleus of the lateral lemniscus inferior colliculi and medial geniculate bodies (Fig 6)

Clinically, lesions in the end organ and the nerve trunk or perhaps the cochlear nucleus (some audiologists prefer "retrocochlear"), which produce a sensory neural hearing loss are differentiated by audiologic tests as follows

| *End Organ* | *Nerve Trunk or Cochlear Nucleus* |
|---|---|
| Partial or complete recruitment | Usually no recruitment |
| Fair discrimination | Very poor discrimination |
| Bekesy Type II | Bekesy Type III or IV |
| Partial Tone Decay | Severe Tone Decay |
| SISI 60–100% | SISI 0 20% |

Lesions in the spiral ganglion are suspected on the basis of laboratory studies however, they cannot be demonstrated clinically The central pathways include a multitude of crossed and uncrossed fibers which connect the cochlear nuclei to the ocular nuclei to account for reflex eye movements, to the nuclei of the fifth and seventh cranial nerves to control tone in the tensor tympani and stapedius muscles respectively, and ultimately to

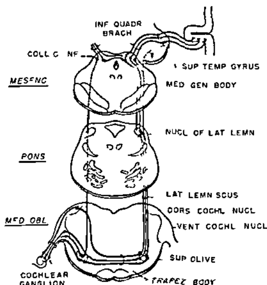



FIG 6 Central cochlear pathways (Brodal)

each superior temporal gyrus so that impulses from each cochlea are projected bilaterally There are also cortico fugal fibers from other areas of the cortex to the nuclei in the acoustic system It follows, therefore, that central auditory pathway lesions cannot be localized by ordinary clinical methods Indeed patients, especially young children, with apparent bilateral sensory-neural hearing loss, may be reacting as they do because of one of four possibilities, two of which are not even primarily concerned with the auditory system

1 Structural disorder of the cochlea, spiral ganglion or eighth nerve trunk
2 Disease or damage in the central acoustic pathways
3 Mental retardation
4 Emotional disturbance

### Eighth Cranial Nerve—Vestibular Division

The vestibular portion of the eighth nerve enters the lower border of the pons with the auditory portion just behind the seventh nerve The cell *bodies of the vestibular division are located in Scarpa's ganglion in the* depth of the internal auditory canal Peripherally, one branch receives impulses from the utricle and the ampullae of the horizontal and anterior vertical canals (Fig 5) Smaller separate branches convey impulses from the saccule and the ampulla of the posterior vertical canal Centrally, the fibers are distributed to four vestibular nuclei, lateral, *medial, superior* and inferior The nuclei are beneath the floor of the fourth ventricle and so are sensitive to pressure from within the ventricle Central connections include fibers to the eye muscle nuclei through the medial longitudinal

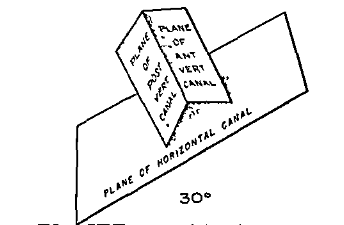



FIG 8 Relationship of the planes of the semicircular canals of one ear to each other and to the horizontal plane The plane of each canal is perpendicular to the planes of the other two The plane of the horizontal canal is at an angle of 30° to the horizontal plane

there are no abnormal sensations However, if equilibrium is disturbed by testing (14) or disease, (28) ocular and spinal responses occur The result of endolymphatic flow has been described above An irritative labyrinthine disturbance produces a slow component to the opposite side It produces the same effect as stimulation with hot water For example, ultrasonic application produces nystagmus toward the affected ear until its function is lost, then nystagmus reverses to the unoperated ear which, with the state of equilibrium upset, acts as an irritated labyrinth Labyrinthine response has been suppressed by repeated exposures to rotational stimulation However, this was attempted in three subjects by daily cold caloric irrigation for two weeks without any change in responses

## Nystagmus

Nystagmus is such an important sign in neuro-otology, that it deserves special consideration There are two general types of nystagmus, ocular (also called pendular), and vestibular (also called jerk) nystagmus

The only concern with ocular nystagmus is that it be recognized and not confused with the nystagmus of vestibular disorder Ocular nystagmus is due to disturbance in fixation associated with loss of central vision found in certain congenital and acquired eye disorders Ocular nystagmus is an aimless wandering of the eyes or irregular oscillation without pattern or rhythm The movements are approximately equal in rate in each direction

Vestibular nystagmus produced by disturbance in the peripheral or central vestibular apparatus has a characteristic jerky rhythm with a slow component in one direction and a fast component in the opposite direction It is frequently accompanied by a false sensation of motion (vertigo) because the displacement of the eyes (13) (slow component of

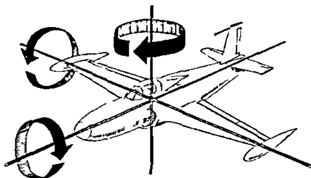


Fig 9 Principal axes of space with relative positions of the semicircular canals Each axis is perpendicular to the other two

nystagmus) is not expected by the cerebral centers and, therefore there is an illusion of motion In voluntary displacement of the eyes the cerebral centers expect the change in eye position and there is no sensation of motion Nystagmus is named according to the direction of the fast component It is described as horizontal, rotary, horizonto-rotary, oblique or vertical depending upon the direction of the fast component It may also be described by degree

First degree nystagmus Nystagmus which is only present when the eyes are turned in the direction of the quick component

Second degree nystagmus Nystagmus which is present when the eyes are turned in the direction of the quick component and also when the eyes are in the mid-position

Third *degree nystagmus Nystagmus which is present in all positions* of the eyes

Opticokinetic nystagmus is a form of vestibular nystagmus produced in normal individuals by looking at a continuously moving panorama The eyes follow the moving object (slow phase) then snap quickly back (quick phase) producing nystagmus in a direction opposite to the moving object. This may be accomplished using a revolving umbrella or drum However, *this is not of practical clinical value*

Fatigue nystagmus, which is always horizontal, is common and of no clinical importance It may occur in a normal person attempting to maintain extreme conjugate gaze beyond the limits of binocular vision (12) It only persists as long as the extreme gaze is maintained

Conjugate gaze is both eyes turned in one direction, right, left, up or down

Conjugate deviation is both eyes fixed in one direction of gaze

Conjugate paralysis is the inability to turn both eyes in one or more than one direction of gaze

Vestibular nystagmus representing disorder in the vestibular *apparatus* may be spontaneous or positional Spontaneous nystagmus is found on

examination without changing head or body position and without any type of vestibular stimulation This nystagmus may be present only with the eyes in the mid position or may be present only on conjugate gaze Positional nystagmus is nystagmus which is produced only by changing position of the head and body (26) Nylen (35) first described two types of positional nystagmus Type I, direction changing, Type II, direction fixed Supposedly Type I is more suggestive of central disease and Type II of a peripheral or central disorder Either spontaneous or positional vestibular nystagmus is diminished in intensity by fixation of the eyes It is, therefore helpful at times to employ Frenzel glasses which are illuminated goggles with 20 diopter lenses which do not permit fixation This allows the examination of a magnified nonfixing illuminated eye

Peripheral vestibular nystagmus is due to disorder in the end organ while central nystagmus is due to disorder in the vestibular nuclei or central pathways Certain characteristics aid in this differentiation for both spontaneous and positional nystagmus

SPONTANEOUS NYSTAGMUS

| *End Organ* | *Central* |
| --- | --- |
| Type | |
| Usually horizonto rotary or rotary<br>Always bilateral | May be in any direction including vertical which is almost pathognomonic of central disorder<br>May be unilateral |
| Course | |
| Most pronounced at onset and gradually subsides over several weeks | Continuous May even increase in severity |
| Associated Symptoms | |
| Severe vertigo<br>Tinnitus and hearing loss | Usually no severe vertigo tinnitus or hearing loss May be other evidences of cranial nerve dysfunction |

POSITIONAL NYSTAGMUS

| *End Organ* | *Central* |
| --- | --- |
| Delay in onset several seconds | No delay |
| Usually horizonto rotary nystagmus (Nylen Type II) | Variable nystagmus (Nylen Types I or II) |
| Severe vertigo | Mild vertigo |
| Limited duration | Unlimited duration |
| Fatigable [illegible] cannot be readily reproduced [illegible] ng) | Non fatigable—may be reproduced repeatedly |

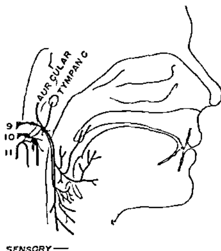



Fig 10 Ninth cranial nerve (Glossopharyngeal)

### Ninth Cranial Nerve (Glossopharyngeal Fig 10)

The glossopharyngeal nerve is primarily a sensory nerve It joins the medulla at the upper end of the posterior lateral portion in line with the tenth and eleventh nerves It leaves the cranial cavity with the tenth and eleventh nerves through the jugular foramen The glossopharyngeal nerve shares the nucleus ambiguus which extends the entire length of the medulla with the tenth nerve and the eleventh nerve The fibers from this nucleus most of which are part of the tenth nerve supply the striated muscles of the pharynx and larynx The inferior salivatory nucleus of the glossopharyngeal nerve supplies glandular structures including the parotid gland and smooth muscle The glossopharyngeal nerve also carries taste sensation from the posterior one third of the tongue and sensation from the adjacent pharyngeal mucosa tonsillar region and soft palate Through its tympanic branch (Jacobson s Nerve) it contributes to the tympanic plexus A supranuclear lesion of the ninth nerve produces no significant symptoms because of bilateral cortical innervation and the fact that it is chiefly a sensory nerve The chief symptom of a lesion of the glossopharyngeal nerve is a loss of sensation in the posterior one third of the tongue pharynx tonsillar region and soft palate This is reflected in a disturbance or absence of the gag reflex This is only clinically significant if it is unilateral

### Tenth Cranial Nerve (Vagus Fig 11)

The vagus nerve joins the medulla just below the ninth nerve The vagus nerve contains most of the fibers from the nucleus ambiguus supplying the musculature of the pharynx and larynx The vagus also has sensory branches from the pharynx and larynx and a branch (Arnold s Nerve) from the skin of the concha and external auditory canal The vagus also

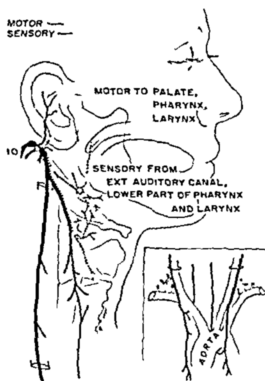



Fig 11 Tenth cranial nerve (Vagus)

contains visceral, efferent fibers which run to the autonomic ganglia of the vagal plexuses The sensory fibers to the pharynx are intimately connected in a plexus with the glossopharyngeal nerve The nerves to the larynx deserve special attention since they may help in neuro-otologic diagnosis The superior laryngeal branch is chiefly sensory and the inferior laryngeal (recurrent) nerve supplies the abductors of the larynx *It is clinically important to remember that the right recurrent nerve passes around the right subclavian artery while the left recurrent nerve passes around the aortic arch*

Because of the bilateral innervation, supranuclear lesions usually do not produce clear symptoms and these symptoms are transient Lesions of the nerve or nucleus produce paralysis of the ipsilateral palate and midline paralysis of the ipsilateral vocal cord If a midline cord is found on examination, the soft palate should be carefully evaluated for paralysis to localize the lesion either above the pharyngeal branches (usually at the jugular foramen or nuclear) or below these branches most likely in the recurrent nerve itself Bilateral paralyses of both ninth and tenth nerves (for example, as found in bulbar poliomyelitis) produce serious problems in swallowing and respiration

### Eleventh Cranial Nerve (Accessory, Fig 12)

The spinal accessory nerve joins the medulla just below the ninth and tenth nerves The spinal accessory affords motor supply to the trapezius

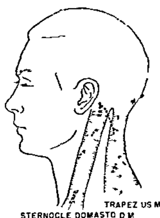



FIG 12 Eleventh cranial nerve (Accessory)

and sternocleidomastoid muscles However it also has autonomic and motor fibers to the pharyngeal and laryngeal musculature connecting with the vagus nerve Lesions of the spinal accessory nerve are uncommon and when unilateral are of diagnostic aid but of little disturbance to the patient

TWELFTH CRANIAL NERVE (Hypoglossal, Fig 13)

The hypoglossal nerve joins the anterior portion of the medulla The hypoglossal nerve is the motor supply to the tongue and also carries proprioceptive sensation from the tongue A peripheral lesion of the hypoglossal nerve produces an ipsilateral paralysis of the tongue so that, when the tongue is protruded the normal muscles on the opposite side push the tip of the tongue toward the side of the lesion Following this atrophy of the tongue occurs with fibrillations Because of the close proximity of the hypoglossal nuclei nuclear lesions are usually bilateral and associated with other serious bulbar disease

CEREBELLUM

The cerebellum which is concerned chiefly with motor co-ordination may be considered a regulating center which maintains graceful balance while standing perfect equilibrium while walking or running and smooth co ordinated movements of all the extremities (4)

Anatomically the cerebellum consists of midline structures collectively called the vermis and a right and left cerebellar hemisphere The vermis includes the flocculo nodular lobe which is the oldest part of the cerebellum Since the cerebellum arose as an out pouching of the vestibular nuclei the vermis is concerned chiefly with equilibratory function that is maintaining proper posture gait and truncal equilibrium The newer portion of the cerebellum the lateral lobes, on the other hand are chiefly concerned

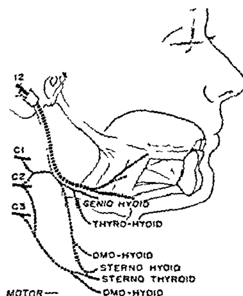



FIG 13 Twelfth cranial nerve (Hypoglossal)

with co-ordination of all the extremities The right cerebellar hemisphere influences the right arm and leg and the left cerebellar hemisphere the left arm and leg

Disorders of cerebellar function manifested by so-called "cerebellar" symptoms and signs may be produced by disease within the cerebellum or disturbances outside the cerebellum which exert pressure on the cerebellum or interfere with cerebellar pathways.

*There are three general rules in cerebellar disease*

1 When symptoms and signs are unilateral they are on the side of the lesion
2 When the midline portion of the cerebellum is affected, symptoms involve truncal function, posture and gait
3 *The more rapid the progress, the more obvious the symptoms and signs* because compensation for loss of cerebellar function takes place so well that slow processes are compensated as they progress and symptoms may be minimal

*Characteristic signs serve to identify cerebellar dysfunction* The otolaryngologist should learn to recognize these signs for, when they are present, the primary lesion cannot be in the labyrinth These signs should be thought of in two groups, those produced by lesions in the cerebellar hemispheres and those produced by lesions in the midline structures

*Signs of Cerebellar Hemisphere Dysfunction (Non-equilibratory)*

1 Asynergia (dissociation of movement) Lack of the proper association and sequence of the contraction of muscles in a compound movement so that the movement is performed in a series of isolated successive movements rather than a single smooth co ordinated movement

2 Dysmetria (past pointing) The inability to control the range of motion so that the mark is either undershot or overshot
3 Adiadochokinesis the inability to perform rapid alternating movements
4 Rebound phenomenon the inability to properly control and contain movement of an extremity after forceful displacement or restraint of the extremity

*Signs of Midline Cerebellar Dysfunction (Equilibratory)*

1 Wide based gait
2 While walking tendency to fall in any direction and the inability to make sudden turns
3 Truncal ataxia while sitting

To summarize In the absence of acute vertigo any one of the above signs indicates that the disorder is in the central nervous system and not in the end organ

# VASCULAR CONSIDERATIONS

Cerebral vascular insufficiency represents either generalized or localized reduction of blood flow to the brain of sufficient degree to impair neurologic function. Temporary inadequacy of blood supply to a localized area of the brain may produce repeated attacks of sensory or motor impairment. These episodes are almost always caused by atherosclerosis in the arteries supplying the brain. It is essential that the otolaryngologist recognize these episodes since they may be early manifestations of an impending cerebral infarction. These attacks may last for a few minutes to several hours and when the vertebral-basilar system is involved, the symptoms may present as labyrinthine disorder.

The blood supply of the brain may be thought of in two systems, the carotid artery and its branches and the vertebral-basilar system (20, 31) (Fig. 14). The vertebral arteries arise from the subclavian arteries and join to form the basilar artery. The vertebral arteries each give off the posterior-inferior cerebellar artery and the basilar artery has the following branches: pontine, internal auditory, anterior-inferior cerebellar, superior cerebellar and posterior cerebral. It is best to think of the vertebral-basilar system as a single system in which deficiency of circulation produces one or a series of symptoms depending upon the ischemic area of the brain or brain stem.

The labyrinth is supplied by the internal auditory artery (Fig. 15) which usually arises from the basilar artery but sometimes from the anterior-inferior cerebellar artery. The internal auditory artery is usually described as an end artery. Actually, such is not the case since the semicircular canals are also supplied by a branch of the stylomastoid artery from the posterior auricular artery. However, this additional supply is not sufficient to supply an effective collateral circulation so that functionally the internal auditory artery may be considered an end artery. The internal auditory artery divides into three branches (3). The first, the vestibular artery, supplies the vestibular nerve and parts of the saccule, utricle and semicircular canals. The second branch is the vestibulo-cochlear artery which supplies the basal turn of the cochlea, the greater part of the saccule, the body and lower part of the utricle, the posterior semicircular canals and parts of the lateral and superior semicircular canals. The terminal third branch, the cochlear artery, enters the modiolus where it gives rise to the spiral arteries which supply the organ of Corti and the stria-vascularis. Venous drainage of the labyrinth is accomplished through three main veins. First,

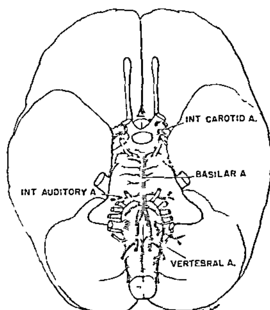



Fig 14 Vertebral basilar artery system

the internal auditory vein drains the apical and middle turns of the cochlea Secondly, the vein of the aqueduct of the cochlea drains the basal turn of the cochlea, the saccule and part of the utricle, and third, the vein of the aqueduct of the vestibule drains the semicircular canals and part of the utricle

The oxygen supply and demand of the brain is unique in that the brain weighing about three pounds, utilizes roughly 15% of the total cardiac output at rest (37) This consumption of oxygen is practically unchanged under such widely different states as the performance of complicated mathematics and deep natural sleep With this maximum use of oxygen,

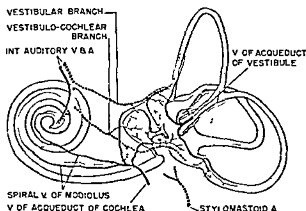



Fig 15 Arterial supply and venous drainage of the internal ear

the brain has a minimum capacity to store oxygen so that continuous maintenance of cerebral blood flow is essential for the survival of brain cells Cerebral blood flow is influenced chiefly by the following factors

1 *Arterial pressure at the brain level*
2 Venous pressure at the brain level
3 Intra cranial pressure
4 The state of the cerebral vascular bed
5 Blood viscosity

Of these, the arterial blood pressure at the brain level and the state of the vascular bed are the most variable and most important The most common cause of vascular insufficiency is atherosclerosis However, there are many other possible causes including cerebral arteritis, cerebral venous thrombosis secondary to infection such as ear and sinus infection, embolism of cardiac origin, systemic hypotension, the aura of migraine, hypertensive encephalopathy, dissecting aortic aneurysm, trauma to the carotid artery and hemotologic disorders

*Episodes of transient focal cerebral ischemia involving the vertebral-basilar system* due to ischemia of the brain stem, temporal and occipital lobes or cerebellum may produce any combination of the following symptoms

1 Sensory or motor impairment unilateral, bilateral or crossed (i e involvement of one side of the face and the opposite limbs)
2 Bilateral blurring of vision or blindness
3 Dizziness or vertigo
4 Dysarthria or dysphagia
5 Paresthesia of circumoral area or tongue
6 Diplopia or nystagmus
7 Nausea staggering gait, ataxia
8 Confusion, faintness or unconsciousness
9 Posterior headache

*In the absence of vertigo or nystagmus, any of the above symptoms* should alert the examiner to the possibility of cerebral vascular insufficiency In addition, it should be emphasized that these focal cerebral ischemic episodes frequently do not last longer than fifteen minutes so that it may be necessary to probe the history very carefully

## HISTORY

History is as important as the physical examination It behooves the otolaryngologist to broaden the scope of his inquiry to include symptoms that if present automatically exclude the labyrinth as the sole problem For example the complaint of transient blindness with vertigo means that the patient must have some central nervous system involvement

The vertigo study questionnaire (Fig 16) has been an invaluable guide in history taking

I Vertigo is the chief common ground between otology and neurology The exact nature of this symptom must be determined True labyrinthine vertigo has the elements of disorientation in space and a sensation of motion Nausea and vomiting accompany or follow severe vertigo when they precede dizziness , the dizziness is probably not true labyrinthine vertigo As a rule the nearer the lesion is to the labyrinth the more true the vertigo Other symptoms (for example lightheadedness swimming sensation pressure sensation) may be unrelated to neuro otological disorder On the other hand a tendency to fall when walking in the absence of vertigo or the association of headache with vertigo suggest central nervous system disease Labyrinthine disorder produces vomiting with nausea while vomiting without nausea occurs in central nervous system disease

II Dizziness which is constant is probably largely functional Most organic vertigo is episodic and/or precipitated by position change Questions as to possible etiology may be revealing For example a history of head injury compels the examiner to test the patient on several different occasions for positional nystagmus A history of drug ingestion (1) may explain spontaneous nystagmus since the use of barbiturates is the most common cause of spontaneous nystagmus In large doses barbiturates may alo produce ataxia Many drugs have been proven to be specifically ototoxic (16 17 19 34 40) and tobacco has been reported to produce destruction of the sensory epithelium (30)

III The importance of complete otologic history needs no emphasis It should however be remembered that the innocent sounding symptom of fullness in the ear frequently represents early serious labyrinthine disease In regard to hearing the examiner should specifically probe for diplacusis which is so characteristic of hydrops of the labyrinth

IV The presence of any of the symptoms in this category suggests central nervous system disease

VERTIGO STUDY — Name Age Case No Date

I When you are dizzy , do you experience any of the following sensations?
*Please read the entire list first Then circle only those which describe your feelings most accurately*

1 Lightheadedness
2 Swimming sensation in the head
3 Blacking out
4 Loss of consciousness
5 Tendency to fall to the right? To the left? Forward? Backward?
6 Objects spinning or turning around you
7 Sensation that you are turning or spinning inside with outside objects remaining stationary
8 Loss of balance when walking Veering to right? To left?
9 Headache
10 Nausea or vomiting
11 Pressure in the head

II 1 Is your dizziness constant or does it come in attacks?
2 How often do attacks occur?
3 When did dizziness first occur?
4 Can you tell when an attack is about to start? Yes No If so how?
5 Does change of position make you dizzy? Yes No
6 When you are dizzy can you stand up unsupported? Yes No
7 Do you know of any possible cause of your dizziness?
8 Were you exposed to any irritating fumes paints etc at the onset of the dizziness?
9 *Do you have any allergies?*
10 Did you ever injure your head?
11 Do you take any medicine regularly? What?
12. Do you use tobacco in any form? How much?

III Do you have any of the following symptoms?
1 Difficulty in hearing? Yes No Both ears right right left
2. Noise in your ears? Yes No Both ears right left
Briefly describe the kind of noise
*Does noise change with dizziness? Yes No If so how?*
3 Fullness or stuffiness in your ears? Yes No If yes in both ears?
In right ear? In left ear? Does this change in any way when you are dizzy? Yes No How?
4 Pain in your ears? Yes No Both ears Right Left
5 Discharge from your ears? Yes No Both ears Right Left
When?

IV Have you experienced any of the following symptoms? If so are they constant or do they *come and go?*
1 Double vision Yes No Constant In episodes
2 Numbness of face or extremities Yes No Constant In episodes
3 Blurred vision or blindness Yes No Constant In episodes
4 Weakness in arms or legs Yes No Constant In episodes
5 Clumsiness in arms or legs Yes No Constant In episodes
6 Confusion or loss of consciousness Yes No Constant In episodes
7 *Difficulty with speech Yes No Constant In episodes*
8 Difficulty with swallowing Yes No Constant In episodes

Fig 16

## PHYSICAL EXAMINATION

The otolaryngologist should organize a systematic examination routine to arrive at a sound clinical impression The following is a suggested order which may be varied according to personal preference

### 1 General Otolaryngological Examination

Complete examination of the nose, nasopharynx, hypopharynx, larynx and tympanic membranes should serve as a base for the entire examination Particular attention should be given to the nasopharynx where obscure tumors may be the primary source of neurological difficulty The examiner should also have a keen eye for paralyses or weaknesses of any of the pharyngeal or laryngeal structures If there is evidence of middle ear infection, the usual methods for demonstrating cholesteatoma and labyrinthine fistula should be employed

### 2 Auscultation of the Skull

Examination for bruits is particularly important in a patient who reports noise in his head Localization of audible noises in the head is important in diagnosis Auscultation of the skull is performed with a bell type stethoscope at six examining points over each eye where sounds are transmitted through the orbital fissures over the squamous portion of each temporal bone which is the thinnest area of the skull, and over each mastoid process where the pulsating tinnitus of a glomus tumor may be appreciated by the examiner Normally, no bruits whatever should be audible in adults

### 3 Examination for Nystagmus

*Spontaneous nystagmus* —The patient is tested in two head positions with the head upright and with the head held back and in the supine position so that the eyes are looking towards the ceiling The eyes are examined looking straight ahead and then in eight positions To elicit horizontal, rotary or horizonto-rotary nystagmus, the patient is asked to look to the right to the left, up and to the right, down and to the right, up and to the left down and to the left To elicit vertical nystagmus, the patient is asked to look directly upward and then directly downward

*Positional nystagmus* —The patient is seated on a table and then drop-

ped back quickly on his back and examined for nystagmus with his eyes in the mid-position There frequently is a latent period of several seconds before the onset of nystagmus If nystagmus occurs, the patient is kept in this position until the nystagmus subsides or if there is no nystagmus, the position is maintained for approximately ten seconds The patient is then quickly raised to the sitting position and again examined for nystagmus The patient is then dropped alternately first on one shoulder then to the upright position, onto the other shoulder and then again to the upright position Each position is maintained for approximately ten seconds unless nystagmus occurs in which case the duration of the nystagmus is noted Throughout this examination, it is important to maintain a constant relationship between the head and body so that the neck is not twisted thus precluding the possibility of neck reflexes influencing the response *The presence of positional nystagmus is evidence of organic disease* (The differences between the positional nystagmus of peripheral and central origin have been described on page 16 ) Vertigo with change of position without accompanying nystagmus may not indicate organic disease

## 4 Examination of the Cranial Nerves

*Fifth Cranial Nerve*

Testing the sensory division of the fifth nerve The corneal reflex is of particular importance in evaluating cerebello-pontine angle lesions The patient is asked to look to one side and then using a tightly wound piece of facial tissue, the examiner approaches from the opposite direction first touching the sclera which should not produce blinking and then slipping the facial tissue onto the cornea which should produce blinking of both eyes If the patient has paralysis of the facial nerve, blinking will not occur on the involved side However, intact corneal reflexes may be demonstrated by producing blinking on the uninvolved side by touching either cornea Cutaneous sensation is examined by comparing the response to pin prick in each division on each side of the face

The motor division is demonstrated by holding a straight object such as a tongue blade on edge in the midline of the face from the tip of the nose to the chin Normally, when the mouth is opened, the chin drops in the midline However if the motor division of the fifth nerve on one side is paralyzed, when the mouth is opened, the chin will be pushed to the paralyzed side by the stronger muscle on the uninvolved side Therefore, the chin will deviate to the side which is paralyzed

*Seventh Cranial Nerve*

The motor division of the seventh nerve is tested in its three major portions 1) the frontalis, 2) the muscles around the eye, and 3) the muscles around the mouth The frontalis portion is examined by asking the patient to wrinkle his forehead Wrinkles should be equal on each

side of the face He is then asked to squeeze his eyes tightly Again the wrinkling should be equal and resistance to opening of the patient s eyes by the examiner should be equal Lastly, the patient is asked to whistle then to produce an exaggerated smile In these two positions wrinkling should be equal In peripheral facial paralysis the entire side of the face is involved, however, in central facial paralysis due to involvement of nerve fibers from the cerebral cortex to the facial nucleus the forehead musculature moves normally since this area receives innervation from both sides of the cerebrum The eyes should be examined for evidence of decreased lacrimation and the tongue tested on each side for taste disturbance This can be accomplished by testing for sweet sour salt and bitter with sugar vinegar, table salt and quinine respectively An estimate of hyperacusis can be gained by holding a well struck 512 cps or 1024 cps tuning fork beside each ear

*Eighth Cranial Nerve—Auditory Division*

Although audiological tests are invaluable in the diagnosis of cochlear and retrocochlear lesions tuning forks should still be a valuable diagnostic tool for the otolaryngologist in his office For tuning fork tests to have any meaning it is important for each examiner to develop his own specific routine so that each patient is examined in the same manner and therefore differences in response will have significance Steel forks have greater reliability than alloy forks The following four tests have been very useful

A *Weber Test* The 512 cps steel fork is placed on the midline of the head or between the clenched teeth The patient is asked if the sound is heard in the center or more on one side or the other In sensory neural hearing loss the fork may be heard louder in the better ear while in conductive hearing loss it may be heard louder in the poorer ear The test is repeated with the 1024 cps steel fork

B *Rinne Test* Using a 512 cps steel fork the patient is asked to compare in each ear air conducted sound with the fork held about one inch from the auricle to bone conducted sound with the stem of the fork pressed firmly against the mastoid process in the region of the mastoid antrum

(1) If air conducted sound is reported as much louder than bone conducted sound in each ear the patient has either normal hearing or a sensory neural hearing loss

(2) If bone conducted sound is reported as louder than air conducted sound in each ear the patient has a conductive hearing loss (In this instance the test is repeated with the 1024 cps steel fork If with this fork the patient still reports bone conducted sound louder than air conducted sound he has a moderate or moderately severe conductive hearing loss )

(3) If air conducted sound is reported louder than bone-conducted sound in the first ear while bone conducted sound is reported louder than air conducted sound in the second ear the test must be repeated on the second ear while masking the first ear If bone conducted sound is still louder than air conducted

sound in the second ear, the patient has a conductive hearing loss in this ear However, if bone conducted sound is no longer heard in the second ear, the patient has a severe sensory neural hearing loss in this ear

C *Bing (Occlusion) Test* If air-conducted sound is reported as louder than bone conducted sound or if they are reported about equal, the patient is asked to occlude the external auditory canal of one ear while it is being tested for bone conduction If the sound becomes louder with the ear occluded, the patient has normal hearing or a sensory neural hearing loss If the sound does not change, the patient has a conductive hearing loss

D *4096 cps Steel Fork Test* After the above tests have been performed, the 4096 cps fork is excited by gentle brushing between the thumb and index finger and held about one inch from the external auditory meatus If the patient has normal hearing or a mild conductive loss, he reports hearing the fork, whereas if he has a significant sensory neural hearing loss, the fork is not heard

*Ninth Cranial Nerve*

Ninth nerve function is tested by evaluating the palatal and pharyngeal reflexes which involve sensation through the ninth nerve and motor response through the tenth nerve The palatal reflex is elicited by gently stroking the soft palate near the uvula and noting equal elevation of the soft palate The pharyngeal reflex is elicited by stroking each side of the posterior pharyngeal wall which produces gagging These reflexes are significant only if there is a distinct difference between one side and the other If the responses are equal (either marked or absent) they are of no diagnostic significance

*Tenth Cranial Nerve*

Total bilateral vagal paralysis produces immediate death Total unilateral paralysis may be noted on examination

With unilateral paralysis, on phonation, the palate on the involved side does not elevate and the palate on the uninvolved side elevates normally The vocal cord on the involved side is in the cadaveric position and remains in this position because the adductors and tensors of the larynx are paralyzed as well as the abductor muscle The pharyngeal reflex of the involved side may be absent If the palate moves normally, but the vocal cord is paralyzed in the midline, the lesion is most likely in the recurrent laryngeal nerve rather than the main trunk of the vagus

*Eleventh Cranial Nerve*

The examiner places one hand on each side of the patient's head and asks the patient to keep his head perfectly straight and resist the attempt of the examiner to move the patient's head The examiner then attempts to forcibly twist the head first in one direction then in the opposite direction As the attempt to twist the head to the right is resisted by the patient, the right sternocleidomastoid muscle becomes very tense, taut and stands

out in relief This indicates good eleventh nerve function This then is compared to the opposite side

*Twelfth Cranial Nerve*

In bilateral peripheral paralysis the tongue lies immobile in the floor of the mouth the patient has indistinct speech and difficulty in eating In unilateral peripheral paralysis with the tongue protruded the tip of the tongue is pushed to the involved side by the stronger muscles on the opposite side Therefore the tip of the tongue in unilateral peripheral paralysis deviates in the direction of the involved nerve

## D CEREBELLAR FUNCTION

### Tests for cerebellar hemisphere function

1 *Asynergia*

Finger to nose test The patient is asked to extend his arm and touch the tip of his nose first with his eyes open and then with his eyes closed The patient is then asked to raise his arms above his head to extend his finger and then bring his arm down and touch the examiner s hand This is repeated with the eyes opened and closed With the patient sitting or preferably lying he is asked to raise his foot and touch the examiner s hand with his toes All of these tests should be performed smoothly gracefully and with accuracy In cerebellar hemisphere disease the movements are gross dissociated and jerky

2 *Dysmetria*

Past pointing can be tested for as described above however the best test is the finger to finger test in which the patient is asked to touch his own nose and then to touch the examiner s finger which is held in front of him The examiner moves his own finger to different positions so that the patient has a new target each time With dysmetria the patient misses the target fairly consistently

3 *Adiadochokinesis*

The patient is asked to rapidly flip his hand on his thigh first striking the palmar surface then the dorsal surface and repeating this rapidly in succession With cerebellar hemisphere disease this cannot be performed quickly and gracefully and a difference between the two sides may be noted

4 *Rebound Phenomenon*

The patient is asked to extend his arm in front of him with his eyes closed The examiner suddenly pushes the patient s hand either up or down Normally the patient s hand snaps back to the original position With cerebellar disease the sudden displacement produces a pendular reaction in the patient s arm so that the arm makes several excursions in each direction before coming to a stop This can also be performed in the lower extremities With the patient lying the examiner holds his hand over the patient s shin and asks the patient

to forcibly raise his leg against this resistance The examiner then suddenly releases the leg Normally, the patient is able to check the rise and stop the leg quickly With cerebellar disease, there is an exaggerated lifting of the leg and some pendular motion

### Tests for midline cerebellar function

Midline function of the cerebellum is examined by first observing the patient in the sitting position to note if he is able to maintain proper posture with ease The patient is then asked to stand with his feet together and his eyes first opened and then closed Again, proper balance should be maintained The patient is asked to hop first on one foot then on the other foot This should be done equally well In cerebellar disease, the patient may be able to perform only on one side The patient is then asked to walk in a straight line and observed for gait and stability. He is then ordered to stop suddenly and turn around quickly This should be accomplished with ease and without any tendency to fall The walking test can be reinforced by asking the patient to walk heel to toe, however, in older patients this is frequently impossible and not a sign of midline cerebellar disease

## 6 Testing of Vestibular Function

There are four basic ways to actively stimulate the labyrinth to evaluate its function These are motion tests, caloric tests, fistula test and galvanic stimulation

The most reliable test for evaluation of a patient in the office is the "mass caloric test" employing water at approximately 68°F which is comfortably cool If the patient has a perforation of the tympanic membrane, cold caloric stimulation can be performed with refrigerated air using the Dundas-Grant tube The purpose of the caloric test is to evaluate the state of irritability of the labyrinth and to aid in differentiating between peripheral and central vestibular disorders

The mass caloric technique allows both quantitative estimation of labyrinthine irritability and qualitative evaluation of vestibular function It has been most useful in differentiating peripheral from central lesions In addition to determining the length of douching necessary to produce labyrinthine response (quantitative estimation), this technique emphasizes the importance of determining the *type* of nystagmus response both from the horizontal and vertical canals (qualitative evaluation) It allows the physician to visually examine for rotary nystagmus and perversions of nystagmus which are highly important qualitative responses for diagnosis The amount of stimulation can be adjusted to allow leisurely examination of responses from the horizontal and the vertical canals separately as well as past-pointing and falling Vertical canal response may afford valuable diagnostic clues The stimulus is not fixed If the labyrinth is less irritable, douching can be comfortably continued as long as necessary If the labyrinth is more irritable, douching can be discontinued sooner Usually nystag-

mus occurs after 20–35 seconds of douching and lasts 3–4 minutes. Decreased labyrinthine irritability is based upon the length of time of douching required to produce a response, not solely on the duration of the response. If there is no induced nystagmus after four minutes of douching, the labyrinth is considered to be non-functioning. In several instances where labyrinths were considered to be non-functioning on the basis of another caloric test method, response was demonstrated by the mass caloric technique douching for up to four minutes. If, before the test, the patient has a spontaneous nystagmus in the direction of the expected response to cold stimulation, the same technique is employed using warm water at 112°F which produces responses in an opposite direction.

1. *Mass Caloric Test*

With the patient sitting, after he has been examined for spontaneous nystagmus and past-pointing, with the head 60° back, one ear is douched with water at 68°F (Fig. 17) until the first jerks of nystagmus are noted. A funnel with tubing attached (Fig. 18) is used so that douching can be continued as long as necessary without difficulty. Nystagmus usually occurs after 20–35 seconds of douching. If nystagmus is not noted, douching is continued until nystagmus occurs to a maximum of four minutes. Once nystagmus occurs, the patient is then examined in two positions, with the head forward 30° which allows maximal stimulation of the vertical canals, and with the head back 60° which allows maximal stimulation of the horizontal canal. Normally, cold stimulation of one ear produces nystagmus to the opposite side. With the head forward (vertical canals stimulated) the nystagmus is rotary, with the head back (horizontal canal stimulated) the nystagmus is horizontal. Accompanying the nystagmus, the patient complains of vertigo and past-points and falls in the direction of the ear which has been stimulated. In addition, depending upon the degree of vertigo, the patient may demonstrate vegetative response with pallor, perspiration, nausea or vomiting.

2. *Cold Air Test*

In the event that the patient has a perforation of the tympanic membrane, cold caloric stimulation may be produced by using refrigerated air. A cone-shaped coil of copper tubing covered with stockinet (Dundas-Grant tube) (Fig. 19) may be used. The coil is first sprayed with ethyl chloride and then compressed air is injected through the tube into the external auditory canal against the tympanic membrane. Air is injected for 15 seconds to produce a response. Nystagmus, past-pointing, falling and vertigo should occur as with the cold water test. Although this test appears rather simple, it is much less pleasant than the mass caloric method, does not easily allow maximal stimulation and is less definitive. Therefore, the cold air test is reserved for patients with a perforation of the tympanic membrane.

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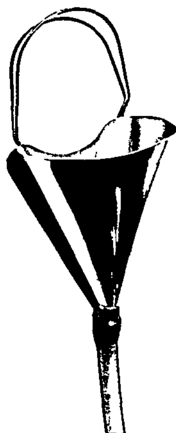


Fig 18 Caloric funnel with tubing attached used for mass caloric test

tion is eliminated In peripheral labyrinthine disease, there is proportionality of response This means that even though the labyrinth may be depressed, depression of vertical and horizontal canal function is equal and the response from each labyrinth is also essentially equal In peripheral disease, regardless of the degree of depression of labyrinthine function,



Fig 19 Dundas Grant tube for air caloric test

the *type* of response is normal This means that stimulation of the horizontal canal produces a horizontal nystagmus to the opposite side while stimulation of the vertical canals produces a rotary nystagmus to the opposite side

*In central vestibular disturbance*, early in the disease, mass caloric test may indicate decreased labyrinthine irritability, while somewhat later in the disease (but still very early when compared to clinical findings) maximal caloric stimulation may produce no response whatever Responses may be disproportionate For example, there may be reasonably normal response from the horizontal canal and essentially absent response from the vertical canals In central disorders there may also be inversion of responses, that is, response in a direction opposite from that which is expected, or perversion of responses, which is nystagmus entirely different from that which is expected In central disorders, conjugate deviation of the eyes may follow caloric testing In posterior fossa disease, vegetative response (pallor, perspiration, nausea, vomiting) is absent

## PRINCIPAL DISORDERS TO BE CONSIDERED

The following disorders present symptoms which may suggest primary ear disease or primary neurological disease and therefore these disorders fall into the realm of neuro otology The following summaries will aid in their diagnosis Where applicable the following disorders will be discussed as to (1) predisposing factors (2) main symptoms (3) signs (4) caloric response (5) audiologic examination and (6) chief diagnostic criteria

1 Meniere s disease (Hydrops of the labyrinth)
2 Cerebello pontine angle mass lesion including acoustic neuroma
3 Glomus jugulare tumor
4 Benign paroxysmal positional nystagmus and vertigo
5 Vestibular neuronitis
6 Vertebral basilar artery insufficiency
7 Labyrinthine ischemia
8 Occlusion of the posterior inferior cerebellar artery
9 Sudden deafness
10 Physical trauma to head with labyrinthine concussion and vertigo
11 Labyrinthitis (inflammatory)
12 Petrositis
13 Brain abscess
14 Hereditary ataxia
15 Vertiginous epilepsy
16 Multiple sclerosis

### 1 Meniere s Disease (Hydrops of the Labyrinth)

Many predisposing factors have been associated with the etiology of Meniere s disease however there has been no universal agreement on any single factor As Williams suggests it may well be a fundamental physiologic disorganization (51) probably significantly influenced by personality factors At any rate it is generally agreed that certain activities or occurrences may precipitate attacks

*Symptoms*

1 Recurrent episodes of severe rotational vertigo with absence of dizziness between attacks

2 Nausea and vomiting accompany severe vertigo
3 Fluctuating hearing loss with diplacusis in that the pitch and quality of the same sound is heard as different in the normal and involved ear.
4 Roaring tinnitus
5 There may be an aura of an attack in that the hearing loss or tinnitus may change in degree or quality just prior to an attack of vertigo

*Signs*

1 Sensory neural hearing loss in the involved ear on tuning fork testing
2 Horizonto rotary nystagmus only during an attack with none between attacks
3 No positional nystagmus
4 No other central nervous system signs

*Caloric test*

Usually responses are decreased, however, they are never absent and there are never perversions of responses

*Audiologic examination*

1 Relatively flat sensory-neural loss for air and bone pure tones
2 Commensurate loss for speech
3 Moderately poor discrimination
4 Recruitment
5 Bekesy Type II
6 Partial tone decay
7 SISI 70–100%

*Diagnosis*

Diagnosis is made on the character of the vertiginous episodes and tinnitus diplacusis, the absence of other central nervous system signs and the audiologic examination

## 2 Cerebello Pontine Angle Mass Lesion Including Acoustic Neuroma

There are no specific predisposing factors other than possibly a family history of von Recklinghausen's disease

*Symptoms*

1 The earliest symptom is usually a gradually progressive unilateral hearing loss associated with tinnitus (8, 45)
2 Gradually progressive unsteadiness and difficulty with balance
3 Episodic true vertigo is usually not an outstanding symptom
4 As the disease progresses, other cranial nerves become involved producing numbness and paresthesia of the same side of the face and ipsilateral involvement of the seventh, ninth, tenth, eleventh and twelfth nerves (29)
5 Also as the disease progresses, marked ataxia and headache may become prominent

*Signs*

1 Early in acoustic neuroma there are no signs other than sensory neural *hearing loss which is only noted if hearing is tested*
2 Diminished or absent corneal reflex
3 In early cerebello-pontine angle tumor numbness of the face may be the first sign (fifth nerve involvement)
4 Persistent nystagmus usually horizontal and more marked toward the side of the lesion and usually most pronounced when the patient is lying Nystagmus may change direction with change of position
5 Cerebellar signs
6 Involvement of other cranial nerves (seven nine ten eleven or twelve)
7 Papilledema may occur later in the disease
8 Elevated cerebrospinal fluid protein

*Caloric test*

1 Early in the disease decreased irritability of the labyrinth on the involved side
2 Also early in the disease there may be perversion of responses from the involved ear
3 Somewhat later in the course of the disease *but still relatively early in* regard to neurological signs caloric response is completely absent in the involved ear and perversions may be noted in the responses from the opposite ear
4 There is no sensitivity reaction (pallor perspiration nausea) to caloric testing

*Audiologic examination*

1 Early in the disease
   (*a*) Moderate pure tone sensory neural hearing loss
   (*b*) Disproportionate very poor discrimination
   (*c*) No recruitment
   (*d*) Bekesy Type III or IV (23)
   (*e*) Severe tone decay
   (*f*) SISI 0–20%
2 Later in the disease there is no response from the involved ear

*Diagnosis*

Early diagnosis depends upon a high index of suspicion in unilateral sensory neural hearing loss with prompt audiologic evaluation caloric test and x ray studies including laminography of the petrous pyramid and contrast studies of the cerebello-pontine angle and internal auditory canal Later diagnosis is made on the other cranial nerve and cerebellar involvement and elevated cerebrospinal fluid protein

## 3 Glomus Jugulare Tumor (7, 41)

*Glomus jugulare tumors occur most commonly in middle age* and much more frequently in women than in men

*Symptoms*

1 Mild hearing loss with or without a sensation of fullness in the ear
2 Pulsating tinnitus which may be described as synchronous with the pulse
3 Aural discharge with or without ear pain
4 Facial paralysis with increased hearing loss
5 Vertigo
6 Later involvement of cranial nerves nine ten eleven and twelve in addition to seven and eight

*Signs*

1 Early conductive unilateral hearing loss
2 Tinnitus which may be temporarily stopped by pressure on the carotid artery
3 Polypoid mass which may bleed easily in the external auditory canal
4 If the drum is intact the tympanic membrane may be discolored and a deep red mass may be appreciated in the middle ear behind the intact drum The mass may blanch when pressure is applied with a pneumatic otoscope
5 Bruit over the ear or mastoid
6 Sensory neural hearing loss with the above signs
7 Facial paralysis and later paralyses of the ninth tenth eleventh and twelfth cranial nerves

*Caloric test*

1 Normal type responses but irritability of the labyrinth may be decreased if there is a large mass in the middle ear Late in the disease with invasion of the labyrinth there may be no caloric response

*Audiologic examination*

Conductive hearing loss early and sensory neural hearing loss later if the labyrinth is invaded

*Diagnosis*

Clinical diagnosis is made on the symptoms of hearing loss and pulsating tinnitus synchronous with the pulse and the appearance of the external canal ear drum and middle ear There may also be a bruit X rays of the temporal bone including laminography, may reveal destruction of the labyrinth mastoid or petrous pyramid Carotid arteriogram may be helpful and retrograde jugularography has been suggested (22) Finally pathologic diagnosis is made on biopsy

*Note*

It should be pointed out that a mass behind the drum as described above may represent a prominent jugular bulb presenting through the floor of the middle ear Therefo [illegible] panotomy is performed for diagnosis in such a case the mass sho[illegible] be aspirated first before performing a biopsy to determine whether or not [illegible] is a solid tumor or the jugular bulb

## 4 Benign Paroxysmal Positional Nystagmus and Vertigo

Usually occurs for no obvious reason but frequently follows head injury (2) Also reported to occur in prolonged illness general anesthesia chronic otitis media and following stapes surgery (42)

*Symptoms*

1 Sudden transient vertigo with change of position frequently only in one position or more severe in one position
2 Infrequent associated hearing loss and tinnitus

*Signs*

1 Severe rotary nystagmus of short duration induced by sudden position change
2 Latency—nystagmus begins several seconds after position has been changed
3 Severe vertigo persists only while nystagmus is present Vertigo subsides promptly as nystagmus subsides
4 Fatigability—nystagmus and vertigo cannot be immediately reproduced with the same intensity With repeated testing nystagmus and vertigo no longer occur however after a period of rest nystagmus and vertigo return with position change

*Caloric test*

Normal

*Audiologic examination*

Not characteristic usually normal

*Diagnosis*

History of postural vertigo with classic signs of positional nystagmus described above and without other neurological or otological symptoms or signs

*Note*

It is recognized and accepted that the description benign may be in error Therefore, it is imperative that every patient with this diagnosis be completely evaluated and re evaluated on several occasions However to date in our experience none of the patients presenting this symptom complex as an initial complaint have been shown or found to have a central disorder (27)

## 5 Vestibular Neuronitis

Associated with infection anywhere in the body either on a toxic or allergic basis (9) In patients under 30 years of age, it is reported to be a more common disorder than Meniere s disease In general, it is usually found between the ages of 20 and 50

*Symptoms*

1 Sudden vertigo which increases in intensity for from several hours to several days

*Symptoms*

1 Sudden onset of severe vertigo
2 Difficulty in swallowing
3 Difficulty in speech

*Signs*

1 Nystagmus of varying direction
2 Signs on the same side as the lesion
   (a) Impaired touch on the face
   (b) Horner's Syndrome
   (c) Cerebellar signs
3 Sign on the opposite side
   (a) Disturbance in pain and temperature sensation

*Diagnosis*

Diagnosis is made on finding of sudden vertigo with the classic neurological symptoms and signs described above

## 9 Sudden Deafness

Occurs in any age group without known cause Essentially the same disease has been described as due to hemorrhage into the labyrinth (labyrinthine apoplexy) (24, 25) and as due to viral infection (15, 43)

*Symptoms*

1 Usually sudden sensation of fullness in one ear
2 True rotational vertigo which continues in diminishing intensity for several days
3 Sudden severe unilateral hearing loss which usually does not improve
4 High pitched tinnitus

*Signs*

1 Severe unilateral sensory neural hearing loss on tunning fork tests
2 Nystagmus during vertigo
3 No central nervous system signs

*Caloric test*

Decreased labyrinthine irritability

*Audiologic examination*

1 Severe sensory neural hearing loss (5)
2 Recruitment (15)

*Diagnosis*

Sudden onset of vertigo and hearing loss which does not improve and without other otologic or central nervous system abnormalities

## 10 Physical Trauma to the Head with Labyrinthine Concussion and Vertigo

Head injury may produce a fracture of the temporal bone or concussion of the labyrinth with or without fracture (38 44)

*Symptoms*

1 Recurrent severe vertigo with change of position for several days to many months following the injury
2 Throughout this time the patient may note a vague sense of loss of balance without sudden position change
3 Hearing loss

*Features of Fracture of the Temporal Bone*

| *Longitudinal Fracture (80%)* | *Transverse Fracture (20%)* |
|---|---|
| Injury to the parietal or temporal region of the skull | Injury to the occipital portion of the skull |
| Usually seen on x ray | 60% seen on x ray |
| Associated facial nerve paralysis less than 25% | Facial nerve paralysis about 50% |
| Laceration of the tympanic membrane with bleeding | Intact tympanic membrane but hemo tympanum |
| Positional vertigo and nystagmus | Severe vertigo at the onset then sub siding vertigo |
| Normal type caloric responses although labyrinthine irritability may be somewhat decreased | Absent caloric response |
| Conductive hearing loss with super imposed sensory neural loss if con cussion was severe | Profound sensory neural hearing loss |

*Diagnosis*

Shortly after the accident if the state of consciousness permits examination diagnosis is made on hearing examination which can usually be performed with tuning forks using adequate masking Later complete audiologic studies and tests for positional nystagmus determine the residual defect

## 11 Labyrinthitis (Inflammatory)

Follows acute or chronic infection of the middle ear or mastoid (50) May also occur in the course of meningitis

*Symptoms*

1 Vertigo which may be aggravated by pressing on the tragus if a labyrinthine fistula is present
2 Unilateral hearing loss
3 Distortion of hearing and diplacusis (46)
4 Usual symptoms of acute or chronic middle ear and mastoid disease

*Signs*

1 Spontaneous nystagmus toward the affected ear in serous labyrinthitis and toward the opposite ear in suppurative labyrinthitis
2 Absence of other cranial nerve disturbances or cerebellar signs unless it occurs in the course of meningitis
3 Fistula test may be positive
4 Usual findings of acute or chronic otitis media and mastoiditis

*Caloric test*

1 Decreased labyrinthine irritability in serous labyrinthitis
2 Absent responses in suppurative labyrinthitis

*Audiologic examination*

1 Mixed loss with increased sensory neural loss greatest for the high frequencies in serous labyrinthitis
2 Total hearing loss in suppurative labyrinthitis

*Diagnosis*

Acute onset of vertigo and increased hearing loss in the course of acute or chronic ear disease without central nervous system symptoms or signs unless they occur in the course of meningitis

*Note*

Diagnosis of suppurative labyrinthitis cannot be made until the acute process has subsided and it has been determined that all vestibular and cochlear function has been destroyed

## 12 Petrositis

Usually follows acute mastoiditis but may occur in the course of chronic mastoiditis

*Symptoms*

1 Persistent pain especially around the eye
2 Persistent or reappearance of aural discharge following mastoidectomy
3 Diplopia
4 Mild vertigo

*Signs*

1 Persistent discharge
2 Ipsilateral sixth nerve paralysis
3 May have seventh nerve weakness on the same side
4 Low grade fever
The occurrence of meningitis during acute otitis media without sufficient a holo y in the mastoid (46)
X ray changes

*Caloric test*

Normal or decreased labyrinthine irritability unless suppurative labyrinthitis is associated

*Audiologic examination*

Conductive hearing loss unless suppurative labyrinthitis is associated

*Diagnosis*

Persistent pain and persistent or recurrent discharge in chronic mastoid disease or following mastoidectomy associated with sixth nerve paralysis on the same side

## 13 Brain Abscess

Occurs most frequently as a complication of chronic suppurative otitis media and mastoiditis with cholesteatoma and rarely as a complication of acute suppurative otitis media and acute mastoiditis Temporal lobe abscess which is due to extension through the tegmen is much more common than cerebellar abscess which is extension through Trautman's Triangle (Fig 20)

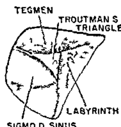



Fig 20 Dissected temporal bone showing the tegmen which separates the mastoid process and middle ear from the middle cranial fossa and Trautman s triangle (bounded by the lateral sinus the tegmen and the labyrinth) which separates the mastoid process from the posterior fossa

### *General symptoms and signs*

*Symptoms*

1 Stage of invasion—chills and fever with headache which may be obvious and last several days in some patients but on the other hand may be very minimal and pass completely unnoticed in other patients
2 Latent stage—no significant symptoms—may last several days to several weeks
3 Manifest stage—rapid progression of increased intracranial pressure with headache nausea and vomiting There may also be focal or generalized convulsions

*Signs*

1 Stage of invasion—moderate fever which may pass unnoticed
2 Latent stage—no characteristic signs

encephalogram did not demonstrate the ventricular system satisfactorily, therefore, a ventriculogram was performed This suggested a mass lesion with displacement of the cerebral aqueduct. Craniotomy was, therefore, performed The cerebellar tonsils were found to be displaced downward There was a tumor on the roof of the fourth ventricle involving the midline structures of the cerebellum and blocking the cerebral aqueduct Pathologic diagnosis was cerebellar sarcoma and the patient died several days following surgery

*Comment* —This case demonstrates how a cerebellar tumor may present a paucity of signs Actually, at one point, a functional disorder was considered This patient demonstrates the value of a caloric test as additional confirming evidence of central nervous system disease in a patient who presented essentially only increased cerebrospinal fluid pressure until a ventriculogram was performed

*Case 3*

A L., 29-year-old male was first examined in February, 1964, complaining of numbness and weakness of the right side of his face associated with dull aching in the right ear, mild hearing loss and fullness in the right ear for the past week He reported that in the summer of 1963, he noted a gradual loss of hearing in his right ear and in the fall of 1963, he developed numbness and weakness of the right side of his face This came on rather suddenly and was not associated with pain However, at that time, he noted an increased sensitivity to loud noises He was treated by his physician and reported that his symptoms disappeared until one week prior to this initial examination He had had occasional mild dizziness but no severe rotational vertigo at any time He never had tinnitus or obvious ear infection There was no allergic history and his general health was reported as satisfactory

Examination revealed a moderate right peripheral facial paralysis, a markedly diminished right corneal reflex and decreased sensation on the right side of the face He also had a mild sensory-neural hearing loss in the right ear on tuning fork examination Mass caloric test was performed and was normal in each ear There was no spontaneous or positional nystagmus, however it was noted that the right pupil was larger than the left In view of these findings, the patient was referred for neurological evaluation The above examination was confirmed and, in addition, it was noted that the pupils did not react to either light or accommodation

The patient was hospitalized for further study On skull x-ray, it was reported that the right internal auditory canal was not as well visualized as the left however, it was felt that the skull x-ray was probably normal Cerebrospinal fluid protein was reported as elevated Mass caloric test was [illegible]ted and again was found to be perfectly normal Audiologic examina- [illegible] reported as follows The left ear was normal There was a mild [illegible] neural hearing loss in the right ear with a dip at 4000 cycles.

Average loss for air conduction in the right ear was 13 decibels left ear 5 decibels Speech reception threshold 16 decibels in the right ear, 10 decibels in the left ear Discrimination 88% in the right ear 100% in the left ear Severe tone decay at 2000 and 4000 cycles was noted in the right ear There was no decay in the left ear SISI test was reported as 5% at 2000 and 4000 cycles in the right ear Bekesy was reported as suggesting decay or fatigue in the right ear when 4000 cps was tested with masking in the left ear It was the audiologic impression that the patient had a retrocochlear lesion on the right side

A craniotomy was performed and the posterior fossa explored All the cranial nerves were identified and there was no evident tumor involving the nerves or either cerebello pontine angle A cerebellar biopsy was taken and there was no evidence of tumor The ophthalmologic consultant diagnosed the pupillary disturbance as Adie s syndrome and of no neurological significance The patient developed recurrent keratitis and therefore a tarsorrhaphy was performed

The patient recovered promptly from surgery however he developed mild cerebellar signs Subsequently in September 1964 he had a complete neurological re evaluation including air studies and angiogram and all of these were non revealing His cerebellar symptoms gradually improved however, the facial nerve weakness and the sensory changes persisted

*Comment* —The initial impression in this patient was a cerebello pontine angle lesion most likely an acoustic neuroma However at the present time the neurologic impression is an intra stem disorder most likely a tumor From the otologic standpoint this reconfirms our belief that in the face of perfectly normal caloric responses the diagnoses of either an acoustic neuroma or an angle lesion large enough to produce sensory changes in the fifth nerve are hardly likely if possible Unfortunately this examiner has not had an opportunity to repeat the caloric test following surgery

*Case 4*

O M 71 year old female examined in February 1964 with a history of difficulty in walking for the previous eight to ten months with a tendency to fall towards the left side During this period she had had vague episodes of dizziness but no discrete rotational vertigo She had noted a progressive hearing loss in her right ear without other auditory symptoms She did not have headache

Examination revealed no significant abnormalities of the nose throat external canals or tympanic membranes Tuning fork testing indicated a profound sensory neural hearing loss in the right ear She had gross truncal ataxia with a tendency to fall towards the right There was spontaneous nystagmus with the head forward The nystagmus was rotary to the right and vertical downward on testing the patient several times With the head back there was horizontal nystagmus to the left Mass caloric test was

both were sacrificed. The fifth nerve was identified and found to be flattened like a ribbon. The patient made a prompt recovery from his surgery and, therefore, two weeks later an anastomosis between the twelfth and seventh nerves was performed.

*Comment.*—This patient presented a rather sudden hearing loss in his right ear approximately two years before definite signs of involvement of other cranial nerves made the diagnosis obvious. In this interval, he had had a complete neurological evaluation which was reported as normal. This case points up the importance of early complete evaluation of relatively sudden unilateral hearing loss. It also demonstrates the classical caloric response in a cerebello pontine angle tumor, namely, totally absent response on the involved side and perverted responses on the opposite side.

*Case 7*

F. R., 56-year-old male presented in June, 1964 with numbness of the left side of the face which had been slowly progressive for the previous six weeks. He had no other symptoms at that time. In addition, he began to notice recurrent mild episodes of dizziness and ataxia with loss of balance and falling to the left. He did not complain at any time of true rotational vertigo. He had no significant hearing loss, tinnitus, ear pain or discharge although he complained of occasional "plugged up feeling" in his left ear for several years.

Otolaryngologic examination was normal except for a scarred, retracted left tympanic membrane. He had mild cerebellar signs in the left arm and leg and some asymmetry of the face in that the left side appeared more flaccid than the right. The tongue deviated somewhat to the left and there was spontaneous nystagmus to the left. Caloric test was performed. There was horizonto-rotary nystagmus from stimulation of the vertical canals of each ear and no sensitivity response (pallor, perspiration, nausea) to the caloric test. This finding of perverted response from the vertical canals and absence of sensitivity suggested a posterior fossa lesion. Audiologic analysis was performed and the results were as follows. The audiometric pattern revealed a mild high-tone sensory-neural loss in each ear. Hearing loss for pure tones—2 decibels in the right ear, 3 decibels in the left ear. For speech 8 decibels in the right ear, 8 decibels in the left ear. Discrimination 92% in the right ear, 96% in the left ear. Type IV Bekesy audiogram in each ear and the SISI test was reported as extreme sensitivity to small increases in each ear at 250 cycles. The audiologic conclusion was widespread cochlear disease bilaterally.

Craniotomy was performed and a brain stem glioma was found.

*Comment.*—This case again emphasizes the importance of considering [illegible] f the face as a possible first symptom of central nervous system [illegible] a patient with this complaint, should have a complete [illegible]gical examination including caloric test. The audiologic con-

clusion, in view of the pathology, is interesting and suggests that in this instance, the Type IV Bekesy perhaps indicated a central disorder and the SISI responses were misleading

*Case 8*

A G, 53-year-old male was first examined in July, 1964 complaining of weakness with staggering and some blurring of vision for several months He also had had what he described as sudden falling spells and frequent headaches usually frontal and usually worse in the morning In addition he noted vague numbness of the left side of his face He did not complain of hearing loss, tinnitus, ear fullness, pain, discharge or vertigo

Examination revealed spontaneous horizontal nystagmus on right and left lateral gaze There were no other significant abnormalities of the ears, nose or throat Caloric responses were normal The Romberg test was positive and the patient could not perform tandem gait without falling

Craniotomy was performed and a pontine astrocytoma was found

*Comment* —This indicates how the caloric test can be non revealing at least early in the course of a pontine tumor and points up the fact that abnormal responses are very significant but a normal response does not rule out an intra stem lesion (Refer to comment on Case 3 ) The absence of abnormal audiologic studies in some cases of brain stem disease is notable

*Case 9*

M T, 56-year old male was admitted to the hospital in early August 1964 because of recurrent difficulty with equilibrium for the previous several weeks He also reported being extremely nervous and a history of hypertension In addition, he had noted recurrent numbness of his right arm associated with profuse perspiration and weakness

Initial examination was surprisingly non revealing Blood pressure was slightly elevated While he was in the hospital, he developed several episodes usually with a pattern of numbness and paresthesia of the right arm and face and nystagmus to the right In one striking episode he developed a fever of 103°F with dysphagia numbness of the right side of the face nystagmus to the right falling to the left ptosis of the right eye and a change of pupillary size in which the right was larger than the left Otolaryngologic examination revealed weakness of the right side of the palate and a midline right vocal cord There was also decreased corneal sensitivity on the right and spontaneous oblique nystagmus up and to the right and horizontal nystagmus to the right on right lateral gaze Caloric responses were as follows The chief abnormalities were from stimulation of the right ear This produced an oblique nystagmus up and to the left from the horizontal canal and no response from the vertical canals In view of the spontaneous nystagmus to the right the left ear was tested with both cold and hot water Both of these produced moderately decreased responses but no distinct perversions There was no sensitivity response

to the test (no nausea, perspiration, pallor). The impression from the caloric test was a right posterior fossa lesion Audiologic studies were performed These revealed essentially normal hearing with a flat curve. Average loss for air conduction in the right ear was 15 decibels and the left ear 17 decibels For speech, 10 decibels in the right ear, 14 decibels in the left ear Discrimination was 100% in each ear There was a Type I Bekesy audiogram in each ear The SISI test reported as 0% in each and there was slight tone decay in each ear at 4000 cps

A retrograde aortic arch study was performed and both vertebral arteries were found to be completely occluded The left at its origin and the right filled only its proximal one-third The patient was treated with anticoagulants and gradually improved

*Comment* —This case presents the picture of episodic focal brain stem symptoms in vertebral-basilar artery insufficiency which led to complete occlusion of the certebral arteries Since a craniotomy was not performed, one can only assume that the patient does not have a posterior fossa tumor If this assumption is correct, it indicates that caloric responses can be abnormal in vascular brain stem disease Here, again, it is interesting to note that the audiologic results were essentially completely normal

*Case 10*

H V, 44-year-old female was first examined on September 3, 1964 complaining of hearing loss in her right ear for the past year She denied any associated tinnitus, vertigo, ear fullness, pain or discharge She had a history of mild respiratory allergy She reported her general health as satisfactory and had no other significant systemic symptoms

Examination revealed no abnormalities of the nose, nasopharynx, hypopharynx, larynx, external canals or tympanic membranes Tuning fork testing indicated a moderate sensory-neural hearing loss in the right ear Tests for nystagmus spontaneous horizontal nystagmus was noted on right and left lateral gaze Positional nystagmus was found with the left ear low This was a horizonto-rotary nystagmus which was not fatigable There was very little associated vertigo The Romberg test was negative and there were no distinct cerebellar signs

Caloric test The right ear was douched with water at 68° for four minutes to elicit a satisfactory nystagmus response After four minutes of douching, the nystagmus produced was still of very poor amplitude although it lasted three minutes From the horizontal canal an upward vertical nystagmus was produced Nystagmus from the right vertical canals was rotary to the left Response from the left ear was essentially normal with an onset of normal type nystagmus in 35 seconds lasting 4 minutes and 15 seconds There was no sensitivity response (perspiration, pallor or nausea)

[illegible]he basis of the caloric response and the spontaneous and positional [illegible]stagmus it was felt that the patient had a right posterior fossa lesion

and therefore, hospitalization was advised However, the patient insisted upon returning to her home for approximately two weeks because of urgent personal matters She was admitted to the hospital on September 14 On admission to the hospital findings were the same and in addition tests for cerebellar function revealed cerebellar disorder She had asynergia and dysmetria with the right arm and leg and on heel to toe walking had a tendency to fall to either side Positional nystagmus was still present and there was some suspicion of a decrease in right corneal sensitivity

Audiologic examination was reported as follows Normal hearing for the left ear and a mild mixed loss for the right ear The curve for the right ear had a small peak at 1000 cps Average air conduction loss 40 db right and 5 db left SRT, no response right and 2 db left discrimination, no response right and 100% left In the right ear only Bekesy equivocal Type IV no recruitment but diplacusis SISI 0%, severe decay over the entire frequency range The audiologic impression was a right retrocochlear lesion Pneumo-encephalogram revealed displacement of the brain stem and a mass in the right cerebellopontine angle Angiogram revealed the same mass which was not vascular

On September 23 a craniotomy was performed and a primary choles teatoma was completely removed from the right cerebello-pontine angle The mass was described as yellow with pearly white areas and measured about 6 cm along the petrous ridge and 4 cm vertically The fifth nerve was stretched about 3 cm from the petrous ridge to the pons and the seventh and eighth nerves were displaced downward To date the patient is making a satisfactory recovery

*Comment* —This patient presented with the innocent appearing com plaint of hearing loss in the right ear The finding of spontaneous nystag mus and the type of positional nystagmus which she demonstrated sug gested a central disorder and this was confirmed by caloric testing This emphasizes the importance of the neuro-otological examination in the office of a patient with unilateral hearing loss without other symptoms

## SUMMARY

Pertinent neurological, otological, and vascular data, with their relevant clinical application, have been presented Although it is fully recognized that sophisticated armamentarium is currently available for more detailed evaluation of neuro-otological problems, the material presented enables the practicing otolaryngologist to face the neuro-otological patient with confidence and to gain an accurate clinical impression in a high percentage of patients With this information, he can then direct the patient to treatment or further examination

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# FILTERED SPEECH AUDIOMETRY

*I*

*Basic Studies with Finnish Speech*
*Towards the Creation of a Method for the Diagnosis of*
*Central Hearing Disorders*

BY

ANTTI PALVA

ACTA OTO LARYNGOLOGICA KARLAVÄGEN 41 STOCKHOLM Ö

Vammala 1965 Vammalan Kirjapaino Oy

*To my Wife and Children*

# CONTENTS

# I. INTRODUCTION

The diagnosis of central hearing disorders by audiological methods has proved, virtually up to the present day, almost overwhelmingly difficult. In the early 1950's the examination technique was largely an application of various pure tone tests, speech audiometry had just been perfected for clinical use in the United States, and its adaptation for different languages followed rapidly. It was generally found that audiometric methods did not play a particularly important part in the diagnosis of central defects, while the clinical picture, especially the neurological status, was decisive (Langenbeck 1952, 1963, Wildhagen 1954).

The pure tone tests available were, however, thought to provide some information on central injuries. In his survey of the audiological examination facilities for central hearing disorders Arnold (1951) suggested that the use of masking noise, the study of auditory fatigue, the determination of recruitment phenomenon and intensity difference limen were of some importance to the diagnosis of central symptoms, as were the use of bilateral tests similar to the Stenger test, and the examination of directional hearing.

Intensive research during the 1950's considerably developed pure tone tests. Although the emphasis in research was on the differential diagnosis of cochlear and retrocochlear defects, audiological examination of central disorders also received considerable attention. Of the pure tone tests the ones employed have been those listed by Arnold, either as described or modified. In recent years interest has been directed mainly to the following methods: electrodermal (Bordley and Haskins 1955, Goldstein 1963) and electroencephalographic audiometry (Goldstein 1963), the reflex reaction time to monaural supraliminal stimulus (Chocholle 1954 b, Maspétiol *et al.* 1960, Rubinstein and Meyersohn 1963, Maspétiol and Semette 1964), the intensity difference limen, also studied with simultaneous pure tone stimulus in the contralateral ear (Chocholle 1957, 1959, Maspetiol *et al.* 1960, 1964), the study of sound localization (Matzker 1957, Walsh 1957, Sanchez-Longo *et al.* 1957, Sanchez-Longo and Forster 1958, Matzker and Welker 1959, Greiner *et al.* 1963), the loudness balance test (Jerger 1960), the auditory adaptation (Brunetti 1961, Hahn 1963, Maspetiol and Semette 1964) and post-stimulatory auditory fatigue (Maspétiol *et al.* 1961, 1963) together with contralateral noise, and the paradoxal lateralization of sound (Maspétiol *et al.* 1963, 1964, Goodman 1963).

Many of these tests have provided interesting results, yet Bocca and Calearo (1963) found that »none of the pure tone tests so far suggested seems to be of any clear use for localization purposes»

The test developed by Lucas (1961, 1962) can be considered a kind of intermediary stage between pure tone tests and speech tests, it makes use of the »transitory phenomena» produced by short, rapidly successive pure tone stimuli created by special equipment A similar test has also been described by Pialoux (1962)

The most important achievements in the diagnosis of central hearing disordes in recent years have, however, been made with speech audiometric methods Speech has been used in different ways as test material, both monaural tests and binaural tests requiring central fusion, summation and integration have been found useful in the diagnosis of various central hearing disorders It is natural that speech tests provide a wider variety of results than pure tone tests, since the interpretation of a complex signal in a central disorder presumably causes considerably greater difficulty than the interpretation of a pure tone signal

Meaningful words have proved to be the most satisfactory speech material in studying central hearing disorders (Bocca and Calearo 1963, Fry 1964) One of the primary conditions for the introduction of such a speech audiometric method is that the person tested understands the material offered without any linguistic difficulty Test material and methods to suit the language concerned have therefore been developed in each country for the diagnosis of central hearing disorders by means of speech tests

No speech test for the diagnosis of central hearing disorders has hitherto existed in Finland Since examination methods based on speech material apparently play a main role in the diagnosis of central defects (Bocca and Calearo 1963), a fairly detailed discussion of the different methods employed elsewhere has been considered necessary in order to find the type of tests giving most reliable information on central defects The author later intends to apply a suitable selected method for central hearing diagnosis to the ordinary Finnish speech audiometry that has been in successful use for over ten years (T Palva 1952), and to modify its test material to suit the diagnosis of central disorders

# II REVIEW OF THE LITERATURE

## A. METHODS OF SPEECH AUDIOMETRY IN THE DIAGNOSIS OF CENTRAL HEARING DISORDERS

Any discussion of speech audiometric diagnosis of central hearing disorders must take into account two factors connected with the understanding of speech First, normal speech comprises such a wealth of informative material that, even if some of it failed to reach the auditory cortex as a result of an organic defect or a disturbance due to technical sound transmission equipment, the remaining elements would provide the information necessary for intelligibility Second, the many synaptic junctions of the hearing pathways ensure that, even though a large number of these synapses failed to function, their failure would not be very detrimental to hearing Matzker (1958) states that even when 30 per cent of the synapses of the brain stem are damaged the patient's hearing ability is in practice normal. Only if the speech material can be made more difficult will defects in discrimination be more easily disclosed The terms used to describe this method are »sensibilized speech» audiometry (Bocca 1957, Tato and Quiros 1960), or »low-redundancy speech» audiometry (Bocca and Calearo 1963)

Even with normal test subjects differences can occur in the results of different examiners merely because of less distinct pronunciation This is clearly observable in American speech audiometry, in which the original recording of Phonetically Balanced (PB) words (Rush Hughes) was considerably more difficult to perceive for normal subjects than the PB material recorded later (Ira J Hirsh) Similarly, in various peripheral disorders the former recording gave clearly lower values of discrimination than the latter (Hirsh *et al.* 1952, Silverman and Hirsh 1955) The influence of peripheral defects on the understanding of sensibilized speech entails certain difficulties in the assessment of central disorders But if peripheral hearing has been found normal, at least as far as bone conduction is concerned, observations on central hearing functions are obviously easier to make

Methods of speech audiometry for the diagnosis of central hearing disorders can be divided into two main groups monaural and binaural In the former, the test material is presented separately to each ear, and the results obtained are compared mutually and with normal material A normal cochlear function, however, is usually required for reliable conclu-

sions In the binaural tests, on the other hand, signals, the same or different, are given to both ears, either simultaneously or at brief intervals In this way the combined effect of both ears is employed, and providing that the peripheral hearing mechanism does not produce appreciable distortion, the summation is definitely of central origin

## 1 MONAURAL METHODS

Indistinct pronunciation of test words, even though it would not reduce discrimination by normal persons, does so in some cases of brain tumour (Goldstein *et al* 1956, Goldstein 1961) If syllables are stressed erroneously when the words are pronounced, or if the words of the test sentences chosen are in illogical order, the result is a higher number of mistakes when persons with certain brain lesions are tested (Bocca *et al* 1957) The prolongation of test sentences or of lists of test words reveals a similar phenomenon (Bocca *et al* 1957, Bocca 1958, Quiros 1960) Simultaneous masking speech, louder than the test words definitely reduces discrimination in certain cases of brain tumour (Bocca 1956)

Tests that are easier to standardize, however, are created if the intelligibility of speech is affected by changing intensity or rate of speech, by interruptions, or by restricting its frequency range

### a) CHANGING THE INTENSITY OF SPEECH

Lafon's (1957 a, 1957 b) method uses words grouped according to their phonetic structure, and mistakes in understanding the different speech sounds are described as a function of intensity

Testing starts at an intensity level of 70 db If the number of mistakes at this level exceeds the normal but is reduced to normal when intensity increases, the concept involved is »liminal distortion» But if, with increasing intensity, the number of mistakes remains unchanged or increases, there is »spatial distortion» Both types of distortion are seen in perceptive deafness The test was originally intended for the calculation of the »social adequacy index», but it has since been used for the examination of central hearing disorders by Greiner and Lafon (1957), Greiner *et al* (1957), Mounier-Kuhn and Lafon (1957), Lafon *et al* (1963) and by Mounier-Kuhn *et al* (1963) They showed that, in cases with an injury of the temporal cortex, the contralateral ear gives a higher distortion index than the homolateral ear Bocca *et al* (1957) achieved similar results using five-word sentences as test material and observing the mistakes upon a change of intensity Sa (1958) also drew conclusions concerning central hearing disorders from a study of phonetic mistakes

### b) CHANGING THE RATE OF SPEECH

If speech material is presented at a rate differing from the normal, certain changes are produced in the discrimination of even normal test subjects When the record or band containing the test vocabulary is run at a velocity differing from the normal there also develops a frequency shift, a fact of some importance when results obtained by this method are considered

The normal rate of speech is considered to be 100—150 words per minute (Cherry 1953, Cherry and Taylor 1954) Intelligibility changes as a function of the rate of delivery as follows when the rate increases by 1 6 times discrimination falls to 54 per cent, and when the rate is reduced to 0 5 discrimination is 17 per cent These values have been obtained using relatively difficult speech material (Fletcher 1929) With the use of easier words the results are very different (Klump and Webster 1961) Continuous speech rate can be doubled before discrimination begins to fall (Fairbanks *et al.* 1957)

In slow played speech a change in frequency may be more important than one in rate (Kurzrock 1956) The lowering of the frequency, in particular, reduces intelligibility sooner than does the raising of it (Fletcher 1929) Largely because of this the intelligibility of the male voice suffers more than that of the female voice (Tiffany and Bennet 1961)

Speech can be presented at a rate differing from normal, even without frequency shift Results obtained by this method show slightly better values of discrimination than the values of the studies referred to above (Garvey 1953)

In connection with central hearing disorders discrimination has been found to fall more than normally when the rate of speech is increased (Bocca 1956, Calearo and Lazzaroni 1957, Antonelli *et al* 1963 a, 1963 b), or when the rate of delivery is varied irregularly during the test (Bocca *et al.* 1957) The reduced discrimination of the time-compressed speech, according to Maspetiol and Semette (1964), is typical for cortical lesions Similar results have also been obtained in presbyacusis (Finzi 1956, Calearo and Lazzaroni 1957)

Sometimes even the ordinary rate of speech is too fast for some central disorders, and discrimination becomes normal only if speech is presented more slowly (Bordley and Haskins 1955)

### c) INTERRUPTION OF SPEECH

If speech is interrupted rhythmically so that the omitted and the remaining part are of equal length in time, interruption does not affect intelligibility so long as the interruption frequency exceeds 3000/sec. Only after the frequency falls below 10/sec does intelligibility fall below 70 per cent If the omission percentage increases, intelligibility is impaired if 25 per cent

of the speech is left, intelligibility at an interruption frequency of 10/sec is about 60 per cent When the proportion of speech falls to 12 5 per cent, intelligibility rises above 20 per cent only after the interruption frequency has increased to over 1000/sec (Bocca and Camisasca 1950, Miller and Licklider 1950)

Bocca (1956, 1958) and Antonelli *et al.* (1963 a, 1963 b) made use of this phenomenon in the diagnosis of central hearing disorders and found that discrimination values were definitely below the normal in cases with a temporal lobe tumour With this method Maspetiol and Semette (1964) obtained positive results in subcortical lesions Similar findings have also been obtained in presbyacusis (Bocca 1956, 1958, Antonelli *et al.* 1963 a, Kirikae *et al.* 1964)

d) FREQUENCY FILTRATION OF SPEECH

The effect of frequency filtration on the intelligibility of speech for normal subjects will be discussed in greater detail later (p 22)

Bocca *et al.* (1954, 1955) were the first to make use of filtered speech for the diagnosis of central hearing disorders They employed a low pass filter with a cut-off frequency of 450 cps and attenuation of 30 db/octave The discrimination of normal subjects was about 60—80 per cent, almost the same for both ears With injuries of the temporal lobe a fall exceeding the normal was recorded, especially in the discrimination of words heard by the contralateral ear, although no signs of abnormality had been noted in the ordinary speech audiometric test

Linden (1960, 1964) used two different frequency bands (560—715 cps and 1800—2200 cps) for frequency distortion in tumours of the temporal lobe he found the same changes as Bocca had recorded with his test

Similar results have also been obtained with low-pass filtration by Hennebert (1955), Jerger (1960), Antonelli *et al.* (1963 a, 1963 b), and by Maspetiol and Semette (1964) in temporal lobe damage, by Jerger *et al* (1960) for patients with parkinsonism of postencephalitic or arteriosclerotic origin, and by Kirikae *et al.* (1964) in presbyacusis Flowers and Costello (1963) found that children with serious speech defects of central etiology had more than the normal difficulty in understanding filtered speech.

## 2 BINAURAL METHODS

Various peripheral disorders can interfere considerably with interpretation of the test in all the monaural methods But if the combined effect of both ears is utilized — which occurs clearly only on the central level — at least some of these peripheral disturbances can be excluded

The simplest method of binaural speech audiometry is to present the test words binaurally and compare the results with those obtained monaurally Because of binaural summation the discrimination increases, with identical intensity increases, slightly more than on monaural presentation Based on this Groen and Hellema (1960, 1963) concluded that a central hearing disorder existed whenever the discrimination values for binaurally presented speech did not differ from those on monaural presentation, or when the articulation curve only shifted towards lower intensities Increased steepness of the binaural curve suggested a peripheral perceptive defect

More sensitive and more specific tests are, however, obtained if even binaurally presented speech is sensibilized by certain methods

a) SWITCHED SPEECH TEST

When speech is delivered in equal periods of very rapid alternation between the ears, each ear receiving half of the speech, discrimination is clearly reduced at a certain rate of alternation

If the alternation is slow, about 1/sec, the discrimination is 100 per cent and the voice is heard alternately in both ears With a switching frequency of 20—50/sec most of the words are understood but the spread of results is very great With these frequencies the test words seem to be heard by both ears simultaneously Between the frequencies of 1/sec and 20/sec there is an area where attention is drawn alternately to one and then to the other ear, but not quickly enough for good discrimination With a switching frequency of 3—7/sec, discrimination falls to 0 per cent (Cherry 1953, Hennebert 1955) The rate of speech, however, affects the results considerably If 85 or fewer words are uttered per minute, such a distinct reduction in discrimination does not emerge, whereas at the normal speech rate this effect is clearly visible (Cherry and Taylor 1954, Calearo 1957 a) The increase of intelligibility after a certain switching rate is probably due to the fact that the test subject instinctively excludes the signal reaching one ear and concentrates on listening to the other (Cherry and Taylor 1954) The obvious similarity of the switched speech curve to the interrupted speech curve supports this assumption

In Bocca's (1961) opinion, however, »a real summation of the two halves of the messages takes place at any switching rate and the dip, when present, only means that probably some unknown synaptic rhythm is temporarily put out of gear when the switching rate is between three and five per second The increasing rate of switching does not prevent summation from coming into action and deficiencies in discrimination must be ascribed to troubles of binaural integration » This theory is supported by the clinical findings in central defects

Hennebert (1955) employed the switched speech test and found that results varied with the age of the test subject, the type of test vocabulary and

the speaker's voice He found, also, that in central hearing disorders of a certain kind the alternation frequency area in which discrimination fell to zero was markedly wider than the normal Bocca (1960), Calearo (1960), Antonelli *et al* (1963 a, 1963 b), Kirikae *et al* (1964) and Maspétiol and Semette (1964) have also used this test in the diagnosis of central hearing disorders, and it has, moreover, proved of value in simulant examination (Calearo 1957 a) To summarise the clinical studies mentioned above it may be said that »the interest of this test — and a factor which probably makes it a means of diagnosis on its own account — is that no discrimination loss has ever been found in isolated pathology of the temporal auditory cortex, while on the other hand characteristic dips in integration are observed corresponding to the lower frequencies of oscillation in some cases of diffuse cerebral pathology, and in a considerable number of disturbances in the brain stem» (Bocca and Calearo 1963)

b) SIMULTANEOUS BINAURAL PRESENTATION OF DIFFERENT TEST WORDS

Broadbent (1954, 1956, 1957) studied the ability of the brain to receive two different speech signals simultaneously The test subject was made to hear a different three-digit series simultaneously with each ear Immediately after the series of digits had been recited, the test subject repeated what he had heard Broadbent found that »spatially separated sound may pass through the perceptual mechanism successively rather than simultaneously Information may be stored temporarily and only later give rise to selective response (attention) The role of immediate memory is very important in recognition of two different verbal messages presented simultaneously »

Using the above principle, Feldman (1960, 1962, 1965) presented different words simultaneously to both ears, and found that if there was a diffuse central lesion, mistakes in the words presented were approximately equal in number for each ear A distinctly greater deficit in discrimination of words uttered to one of the ears suggested a disturbance of the contralateral temporal lobe The same result was reached by Kimura (1961 a, 1961 b), using Broadbent's technique to examine patients who had been subjected to temporal lobectomy

A test similar in fundamental idea but more fully developed especially for the diagnosis of perceptive hearing disorders was advanced by Katz (1962) He used spondee type words in the following way »The staggered spondaic word test (SSW-test) is composed of 40 items Each item is made up of *two spondaic words such as »upstairs» and »downtown» One spondee is* presented to each ear in a partially overlapping fashion That is, the second monosyllable of the initial spondaic word and the first monosyllable of the second word are transmitted simultaneously to opposite ears Each ear receives an initial word in turn The test provides competition, or

concurrent stimuli in the two ears, for the monosyllabic words trials »stairs» and »down», while trials »up» and »town» are presented normally in a non-competing fashion»

Katz *et al.* (1963) published results on the SSW-test with normal subjects and with subjects suffering from conductive deafness both groups showed very good discrimination »for competing and non-competing trials» But in unilateral cortical defects they found a considerably increased number of mistakes especially in the »competing» words presented to the contralateral ear The difference, compared with discrimination for the »non-competing» words, was very pronounced In peripheral perceptive defects, however, discrimination was reduced for both groups of words in accordance with an ordinary speech audiogram

c) BOCCA'S BINAURAL TEST

In 1955 Bocca proposed a new type of test to examine binaural summation and integration In his method the test words were fed via two separate channels, in Channel 1 the intensity of test words could be varied, while in Channel 2, in addition to an intensity regulator, there was a low pass filter with a cut-off frequency of 500 cps Through Channel 1 the test words were presented at an intensity at which discrimination did not exceed 40 per cent The intensity was then about 5 db below speech reception threshold The other ear received the sound from Channel 2 at an intensity of some 45 db above the speech reception threshold though with the filtration the discrimination did not exceed 50 per cent

When the test words were presented, in the conditions described above, to one ear from Channel 1 and to the other from Channel 2, discrimination in normal cases amounted to the sum of monaural discriminations But when both channels were combined and the test was given either monaurally or, in a free field, binaurally, the louder sound completely masked the weaker, and discrimination did not exceed 50 per cent This showed that summation is of central origin Bocca recommended the test for the diagnosis of central hearing disorders and reported having obtained pathological results in cases of temporal lobe tumour Similar findings have been reported by Calearo (1957 b) and by Jerger (1960) Flowers and Costello (1963) obtained positive results with this test on children who had serious speech defects of central origin

d) MATZKER'S BINAURAL HEARING SYNTHESIS TEST

Fletcher (1929) mentioned Arnold's observation that »the brain is able to combine the sounds obtained from the two ears to complete the proper picture» Arnold had arrived at this conclusion in a test in which speech could be divided by filtration into two parts so that sounds lower than

1000 cps came from one channel to the left ear and those over 1000 cps from the other channel to the right ear. He found that »when speech was transmitted over such a system, there was apparently no distortion produced, although if either one or the other of the two receivers were away the speech was very distorted, and it was hard to recognize what was being said.»

Arnold's observation remained, however, purely theoretical in character, for it took nearly 30 years before Matzker (1956), working from a similar basic proposition, developed a binaural hearing synthesis test for the diagnosis of cerebral hearing defects.

The principle in Matzker's binaural test was to present to each ear separately a completely different part of speech in the form of a given narrow band of the frequency area to be integrated by the brain. He himself described his technique as follows (1959):

»The narrow band-pass filters, a low one from 500 cps to 800 cps and a high one from 1815 cps to 2500 cps, are used and speech signals are transmitted through both of these. Each band itself is too narrow to allow recognition of the test words. However, when both bands are presented together, adequate recognition is possible. The bands are now separated in such a way that the right ear receives only the low-frequency band, while the left ear receives the high-frequency band. Each ear is presented with only one phonetic fragment of each test word, and yet there is excellent integration of the two *fractions indicated by a near* optimal score when the test is given to normal hearing persons.

»Normal recognition of the test words appears to indicate good functioning of the synaptic connections in this region. If, however, the discrimination score is below normal, the synaptic function within the brain stem is judged defective.

»The actual testing is arranged as follows. The patient is first acquainted with the requirements of binaural hearing. This is achieved by letting him answer ten carefully phrased questions transmitted to him through a set of earphones. Next, the patient listens to a series of 41 two syllabic words (phonetically balanced) which are transmitted in binaural fashion as described above. Mistakes are judged only from the phonetic standpoint whereby importance is placed upon the correct recognition of the vowels. The first test is followed by a second one, again a series of 41 words which are phonetically balanced. This series is given so that both frequency bands are received in either ear, that is, in a diotic manner of presentation. Each ear now receives both phonetic fractions together so that the brain stem is relieved of its task of binaural fusion. As a rule, the words are now well recognized.

»Upon completion of the second test, a third list of phonetically balanced words is presented, again in the same binaural fashion as in the first test. This is done in order to force once more bilateral integration at the level of the brain stem. A patient with normal brain function makes

now even noticeably fewer mistakes than in the first binaural test run In contrast, patients with brain lesions make a high score of mistakes, definitely higher than in the second (diotic) run because of their failure of bilateral integration. Normal test results, expressed as the number of mistakes in the three test runs (binaural — diotic — binaural) may look as follows 9—4—3 14—6—6, 12—3—4 In contrast the following results are indicative of pathological changes 27—4—12, 31—7—18, 25—4—13, 37—24—38 It goes without saying that each examiner must gain experience until he is able to tell mistakes and to judge the test results properly »

Having examined over a thousand patients and normal subjects (1959) Matzker noted pathological findings most clearly in cases of brain tumour, irrespective of the site of the tumour the binaural test was positive in 80 per cent of such cases Cerebral atrophy of various origin also usually produced the same effect Hypertension, all age groups included, gave a positive finding in nearly 50 per cent of cases A positive finding was frequent for multiple sclerosis and the condition following a skull trauma

Matzker attributed all pathological findings to a disturbance at brain stem level. His reasoning in the form of pathologico-anatomical findings etc, is presented in a monograph published in 1958 His results were supported by Groen's (1963) negative findings on hemispherectomized patients in Matzker s binaural test

Linden (1960 1964) modified Matzker's test by combining it with a monaural test with filtered speech and running the two tests at four different intensities, when three curves reminiscent of the ordinary speech audiogram were obtained Both bands (560—715 cps and 1800—2200 cps) were first presented at different intensities to one ear, and then to the other and finally both bands bilaterally The words were recorded as a mistake unless they were repeated absolutely correctly This eliminated the examiner's subjective assessment, which exists as a drawback in Matzker's test. In Linden's series there were 18 verified local central lesions usually tumours with either cerebral cortex or brain stem involvement. The result of the binaural test was classified as positive when discrimination was poorer than that of either ear on monaural test. All 18 patients of the series had a negative binaural test, while the monaural test gave positive findings in full keeping with the results obtained by other workers with monaural filtered speech

Matzker's test has also been used to study the central effects of drugs (Rosenau 1962)

R Harris (1963), who examined children with congenital cerebral lesions, modified Matzker s binaural test by using filters to produce two octave-wide bands of each test word. Words were presented alternately so that one word was given, filtered in two bands, bilaterally and the next, with one band to each ear Harris found that »this avoidance of consecutive misperception was important in order to guard against the subject's becoming discouraged or in some way upset by his errors »

frequency was 150 cps, is shown in Fig 1 The test sentence read »Joe took father's shoe bench out, she was waiting at my lawn»

The fundamental frequency of the voice, especially when vowels are pronounced, varies considerably from one person to another The fundamental frequency of a low male voice may be about 90 cps, while a woman with a high voice may speak at fundamental frequency of 300 cps On average, the basic female voice corresponds to $C_1$ or 256 cps, whereas the male voice is about an octave lower (Fletcher 1929)

In vowels the energy is concentrated mainly on the harmonic sounds of the fundamental frequency, which for each vowel are differently divided into typical frequency areas, formants In addition to the fundamental frequency ($F_0$), four formants are usually recognized, the lowest two ($F_1$ and $F_2$) are stronger than the others and occur at frequencies typical of each vowel. The weaker, high-frequency formants ($F_3$ and $F_4$), remain practically constant when vowels are pronounced, in Finnish $F_3$ is 2800 cps and $F_4$ 3280 cps (Wiik 1961)

If the fundamental frequency is raised by an octave the formant values increase by only 17 per cent (Peterson and Barney 1952)

$F_1$ and $F_2$ of the Finnish vowels, according to Sovijarvi (1938), lie at the frequencies indicated in Table 1 The results obtained by Wiik (1961) and by Kytta (1964) agree well with these

The consonants differ essentially from the vowels in that they usually have no distinct formant composition, they are composed of different, mostly high-frequency, noise components In most consonants, however, energy can be found to be concentrated mainly on typical frequency areas For the energy distribution of Finnish consonants, investigation results published by T Palva (1958 a, 1958 b), Sovijarvi (1959), and by Ylppo and Sovijarvi (1962) are available Table 2 shows the main features of the energy distribution of consonants in the Finnish language Consonant *h* is excluded

TABLE 1

*Position of Finnish vowel formants F1 and F2* (after Sovijarvi 1938)

| | F1 (cps) | F2 (cps) |
|---|---|---|
| a | 630—890 | 960—1130 |
| e | 255—510 | 1800—2340 |
| i | 255—450 | 2240—2550 |
| o | 450—465 | 640— 895 |
| u | 360—450 | 600— 645 |
| y | 255—425 | 1665—1800 |
| a | 630—860 | 1345—1900 |
| o | 405—540 | 1520—1730 |

from the table as its own energy is very small and energy distribution depends very much on the sounds surrounding it

Fant's (1948) results of the composition of different sounds are given in Fig 2 Although the study was carried out on the Swedish language the graph gives an immediate overall picture of relations in the frequency area as a function of intensity

TABLE 2

*The main energy distribution of Finnish consonants* (after Sovijarvi 1959 and Ylppo and Sovijarvi 1962)

| | Lower energy region (cps) | Higher energy region (cps) |
|---|---|---|
| d | 100— 200 | 1500—2800 |
| j | 250— 500 | 2100—6800 |
| k | | 900—2250 |
| l | 400—1200 | 1700—3200 |
| m | 200— 250 | 1300—1600 |
| n | 200— 250 | 1300—3450 |
| ng | 200— 250 | 1300—3450 |
| p | 360— 720 | 1120—2850 |
| r | 250—1800 | 2050—4850 |
| s | 250—2100 | 2500—8000 |
| t | 250— 500 | 700—2800 |
| v | 200— 500 | 1400—8000 |

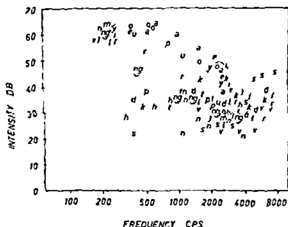



*Fig 2* Frequency composition of the most important vowels and consonants measured with male voice, from Swedish test words at a distance of 1 metre from the speaker's lips (0 db = 0 0002 dyn/sq.cm at 1000 cps) Of the Swedish sounds, only those corresponding most closely to Finnish sounds are shown The areas encircled by thin lines *indicate* the most important formant areas of the vowels, from the left, $F_1$, $F_2$ and $F_3$ (after Fant 1948)

## C EFFECT OF FREQUENCY FILTRATION ON INTELLIGIBILITY OF SPEECH

No results concerning the effect of frequency filtration on the intelligibility of Finnish speech are as yet available The best-known studies in the field are those which have been carried out in the Bell Telephone Laboratory since the 1920 s, especially those by Fletcher, and French and Steinberg, their material consisted of test words in English or of meaningless syllables Fletcher published the results of these studies (1961) reduced to mathematical formulae The selection of test material essentially affects the post filtration results in all studies, the more difficult the test material the lower the articulation percentage obtained with any one filter position

Studies on the effect of filtration on the intelligibility of speech have been conducted mainly with either low pass or high pass filtration, or a combination of both of these, in this way it has been possible to modify at will the width of the bands obtained In many studies, furthermore, background noise has been employed to make the test more difficult and, in particular, to simulate disturbances common in the telephone communication system Although the employment of background noise is not part of the present subject, such studies are described here because their results are also applicable to studies carried out without noise

### 1 LOW-PASS AND HIGH-PASS FILTRATION

French and Steinberg (1947) used meaningless syllables as word material in their studies and examined their intelligibility after low pass and high pass filtration They found that when intensity was increased discrimination improved up to a certain limit, after which it remained largely constant even if intensity was further increased Optimal intensity with different filter positions proved to be approximately the same, within a range of 10 db They considered the »1000-cycle orthotelephonic response» + 10 db (Inglis 1938) of the testing system to be the optimal intensity This corresponds to roughly 75 db relative to 0 0002 dyn/sq cm Using this optimal sound intensity they found that the low pass and high pass curves intersected at 1900 cps, where the articulation percentage was 68 (Fig 3) The curves of Fig 3 are mean values of tests effected with both male and female voice, if male voice alone is employed, the curves intersect in a slightly lower frequency area at about 1660 cps (Steinberg 1929, Miller 1951)

The type of speech material used in the test distinctly affects the intelligibility of filtered speech Meaningless syllables and monosyllabic words suffer most in intelligibility on frequency filtration (Bocca and Pellegrini 1951, Hirsh *et al* 1954), whereas continuous speech tolerates *e g* low-pass filtration up to the cut off frequency of 540 cps, before discrimination falls below 50 per cent (Giolas 1959) The results achieved by Hirsh and his co-workers on

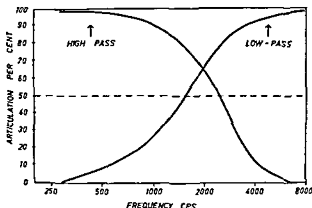



*Fig 3 Effect of low-pass and high-pass filtration on the discrimination of meaningless* syllables using, for each cut-off value of the filter, the optimal intensity +10 db 1000-cycle orthotelephonic response (after French and Steinberg 1947)

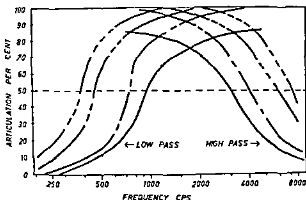



Fig 4 Effect of different word material on discrimination scores obtained with low-pass and high-pass filtration Continuous lines indicate the articulation curve of the meaningless syllables Broken lines indicate the articulation curves of various words, the number of short dashes equalling the number of syllables in the word (after Hirsh *et al* 1954)

different test materials are seen, in idealized form, in Fig 4 In this work a constant 95 db over-all sound pressure level was employed in all test conditions, without modifying the »input» according to filtration The sound pressure level per cycle therefore remained constant in all test conditions. For meaningless syllables the filtration curves are seen to differ slightly in shape from the curves of French and Steinberg (Fig 3), and their intersection is at a slightly lower point, at about 1700 cps The articulation percentages, too, are slightly higher in the study by Hirsh and his co-workers Curves obtained with other types of words, especially polysyllabic words, differ surprisingly from the basic curve of meaningless syllables For example, when high-pass filtration with a cut-off frequency of 6400 cps was used, intelligibility of polysyllabic words was as high as 50 per cent

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results, whereas the mistakes resulting from high pass filtration are more indefinite (Miller and Nicely 1955)

The *elimination of high tones affects the intelligibility of consonants* more than that of vowels, whereas the absence of low frequencies produces a heavier drop in the discrimination of vowels (Fletcher 1929) Wagner (1954) presented a relatively detailed table of the change in the intelligibility of different sounds as a result of low pass filtration According to this table, consonants suffer even on fairly small distortion, first S and F, followed by M, N and Ng whose discrimination is already very difficult when the *cut-off frequency is about 2500 cps Most resistant to distortion are* R, K, T and P Of the vowels, E and I are first affected — they are already easily heard as O and Y or U and O when the cut-off frequency is about 2200 cps A and U best tolerate low pass filtration Lambert's (1954) results concerning the discrimination of consonants are on the same line as Wagner's

In the assessment of discrimination of individual sounds it should be borne in mind that intelligibility often depends decisively on the sounds *preceding and succeeding the sound in question (Bocca and Finzi* 1953 Wang and Fillmore 1961) This is best observed vis a vis consonants (Fry 1964)

## 2. BAND-PASS FILTRATION

*For the theoretical calculation of the intelligibility of* speech bands of varying width, French and Steinberg (1947) used meaningless syllables to divide the frequency area of speech into 20 contiguous bands, all of which had the same effect on intelligibility These bands are listed in Table 3 French and Steinberg maintained that each of the bands added 5 per cent to intelligibility, irrespective of the position of the band Miller (1951) simplified the matter slightly by combining the bands in pairs (1 + 2, 3 + 4, and so on), the result was 10 bands, each providing 10 per cent *intelligibility* Miller plotted a curve (Fig 5) which directly reveals the intelligibility of any given band, provided the number of the »standard bands» it *includes is known*

For the foregoing to be so simply true, the bands should be equal in intensity and the *filter attenuation should* be perfect in other words, no frequencies beyond the cut-off frequencies should be included Furthermore, the test should be carried out without background *noise* which masks different frequencies of speech differently

The effect of band intensity on intelligibility can be summarily assessed if the intensity is 30 db above a threshold value measured in quiet, or a masked threshold value the band concerned gives its maximum addition to intelligibility If the intensity of the band is 16 db the addition to intelligibility is half the maximum and if intensity is 6 db the addition is only one-fifth (Miller 1951)

TABLE 3

*Widths of bands giving the same contribution to intelligibility of speech at optimal sound intensity* (after French and Steinberg 1947)

| Band | Frequency limits cps | Band | Frequency limits cps |
|---|---|---|---|
| 1 | 250— 375 | 11 | 1930—2140 |
| 2 | 375— 505 | 12 | 2140—2355 |
| 3 | 505— 645 | 13 | 2355—2600 |
| 4 | 645— 795 | 14 | 2600—2900 |
| 5 | 795— 955 | 15 | 2900—3255 |
| 6 | 955—1130 | 16 | 3255—3680 |
| 7 | 1130—1315 | 17 | 3680—4200 |
| 8 | 1315—1515 | 18 | 4200—4860 |
| 9 | 1515—1720 | 19 | 4860—5720 |
| 10 | 1720—1930 | 20 | 5720—7000 |

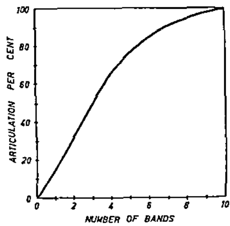



Fig 5 Relation between the discrimination score and the number of standard bands passed by the communication system (after Miller 1951)

Using meaningless syllables, Egan and Wiener (1946) studied the intelligibility of bands of varying width, frequencies of about 1100, 1500 and 2000 cps were employed as center frequencies (geometric means of the cut-off frequencies) of the bands Two male speakers uttered the test syllables at an average over-all level of 70 db (re 0 0002 dyn/sq cm) measured at 1 metre from the speaker's lips Background noise was white noise of an over all sound pressure level of 84 db with a constant spectrum, or a noise of an over-all sound pressure level of 90 db, whose intensity fell however, by an average of 15 db per octave towards the high frequencies

As a result of background noise the intelligibility of the bands, irrespective of their width, was poorer so long as orthotelephonic gain was below + 10 db At this intensity level, intelligibility of even the widest bands was only 20 per cent When the orthotelephonic gain was raised, discrimination improved up to the intensity level of + 30 — + 40 db, subsequent additions to intensity no longer improved intelligibility These results agree with those of Pollack (1948 b) described earlier, for both high-pass and low-pass filtration

From the point view of the present study, the intelligiblity values of the narrowest bands of Egan and Wiener are particularly interesting When the band was 870—1500 cps (center frequency 1100 cps), intelligibility did not exceed 25 per cent even with the optimal orthotelephonic gain A similar low intelligibility was reached for bands of 1300—1900 cps and of 1800—2500 cps (center frequencies 1500 and 2000 cps, respectively) The width of the last two bands was thus about half an octave, and that of the first three-quarters of an octave With bands about one and a half octave wide, there was about 50 per cent intelligibility When meaningless syllables were used, intelligibility remained at only 80 per cent even with bands as wide as 340—3900 cps and 550—6500 cps, which gave 100 per cent discrimination with easier word material

Maspétiol and Semette (1956), using French disyllabic phonetically balanced words, studied the intelligibility of 1/2 and 1/3 octave bands, using 250, 500, 750, 1500, 2000, 3000 and 4000 cps as frequency centres Fig 6 shows their most essential results on using the 1/3 octave band With 1/2 octave bands a similarly shaped curve was obtained, but its apex was at the 80 per cent intelligibility level Judging from these curves the most important frequencies from the point of view of speech intelligibility are about half an octave above and below 1500 cps This agrees fairly well with the earlier studies on the importance of different frequencies for intelligibility

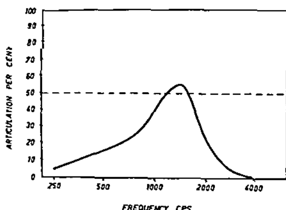



Fig 6 Discrimination of disyllabic, phonetically balanced French words, measured by 1/3 octave wide bands at different frequency areas (after Maspetiol and Semette 1956)

of speech Fowler (1942, 1947) defined the proportion of 500, 1000, 2000 and 4000 cps in intelligibility at 15, 30, 40 and 15 per cent and Fletcher (1950) at 10, 40, 40 and 10 per cent The estimate by Bocca and Pellegrini (1950) was still closer to the results recorded by Maspétiol and Semette with the following percentages 25, 30, 40 and 5

Since the kind of phonetic material employed in the tests essentially affects the results, and since an accurate measurement of the test conditions may sometimes be difficult, employment of ready made formulae (French and Steinberg 1947, Fletcher and Galt 1950, Miller 1951) for the scoring of articulation percentage is often impossible, and intelligibility must be defined experimentally If two or more bands from different sections of the frequency area are presented, scoring of articulation is particularly difficult Pollack (1948 b), for example, concluded as a result of his studies that »the contribution to intelligibility of a given band of speech frequencies is not independent of the contribution being made at the same time by other bands of frequencies Rather there is an interaction among the contributions of the various bands» If, for instance, two separate bands are used, the intelligibility values obtained are very largely dependent on the formant structure of the word material used If the bands coincide with the most important formants ($F_1$ and $F_2$), good discrimination is to be expected If one or both formants remain outside the bands, discrimination is definitely poor

In view of the present study these facts are of very great importance for the selection of bands in a binaural test like Matzker's Only by trying out several different parts of the frequency area can the best possible conditions be achieved with the aid of suitably selected test material

## D HEARING OF BINAURALLY PRESENTED SPEECH

The best known difference between monaural and binaural hearing is sound localization ability which, according to current opinion, is largely based on a small difference in phase arising when sound reaches the two ears at slightly different times (Deatherage *et al* 1959, Deatherage and Hirsh 1959) Binaural hearing gives the sound a three-dimensional effect, while head movements and echoes contribute towards sound localization on a vertical level and towards assessment of distance (Wallach 1940, Licklider 1951, Christian and Roser 1957)

For binaurally effected speech tests, however, there is another important central hearing occurrence that deserves more attention This is the binaural summation phenomenon, on account of which the audibility of monaurally heard sound differs from that heard binaurally

Almost a century ago LeRoux (1875) had already claimed that the addition of supraliminal sound to one ear made a formerly subliminal sound in the contralateral ear audible Observations suggesting that the binaural threshold value was better than the monaural were advanced in the 19th

century by e g Tarchanow (1878) and Urbantschitsch (1893), and later by numerous authors Hirsh (1948 c) has published a good summary of the studies in this field up to that time

Examination of binaural summation is hampered by the fact that the threshold values of the two ears usually differ by some 3—6 db, and this affects the amount of binaural summation (Hughes 1938, Shaw *et al* 1947)

Most authors hold that binaural summation improves the threshold by about 3 db (Hughes 1938, Kemp and Robinson 1937, Causse and Chavasse 1942, Shaw *et al* 1947, Pollack 1948 a, Robinson 1961) Readings range from 1 db (Gage 1932) to 6 db (Hirsh 1948 c) If the frequency gap between the signals presented is widened, the amount of binaural summation is reduced (Bloch 1893, Bekesy 1929, Hughes 1940) According to Hirsh (1948 c), summation is greater with low (below 1000 cps) than with high sounds Chocholle (1954 a) suggested, however, that the amount of summation is independent of frequency

A similar summation phenomenon is also demonstrable with sounds higher than the threshold value It was first noted by Seebeck (1846), and his observations have been corroborated by numerous authors who have also tried to determine quantitatively the amount of binaural summation According to Caussé and Chavasse (1942), summation grows from the threshold value (3 db) with increasing intensity, reaching 6 db at the sensation level of 35 db, after which it remains unchanged Fletcher and Munson (1933) quoted still higher values 12 db at a sensation level of 60 db The general view is, however, that the amount of binaural summation equals that of physical summation (Hughes 1938, Kemp and Robinson 1937, Robinson and Whittle 1960), although no determination of an accurate decibel value has found general acceptance

Binaural summation of audibility holds good in a silent environment or in noise of an intensity not exceeding 50 db per cycle With loud binaural noise, the difference between monaural and binaural audibility depends on the phase relations of noise and test sound if the noise and the test sound are in one and the same phase, binaural audibility is reduced below that of monaural audibility through interaural inhibition If the sounds are in opposite phases, summation occurs These two phenomena are best observed with low sounds not exceeding 1000 cps Interaural inhibition may reduce the binaural threshold of perceptibility so that it is 8 db below the monaural, whereas summation may improve it by about 6 db Hence the effect of phase difference may amount to no less than 14 db (Hirsh 1948 b, 1948 c, Hirsh and Pollack 1948)

The fact that the binaural intensity difference limen is better than the monaural can be attributed to binaural summation (Upton and Holway 1937) The same applies to the frequency difference limen (Shower and Biddulph 1931)

The difference between monaural and binaural audibility is greater when pure tone is employed for the test than if white noise is chosen (Pollack 1948 a).

All the results described above are, in one way or another, applicable to the hearing of speech. Using speech as test material it has been found that binaural summation improves the speech reception threshold just as much as it does the pure tone threshold, in other words, about 3 db (Keys 1947, Shaw *et al.* 1947, Carlo and Brown 1960, Lochner and Burger 1961). In the same way as with pure tones, the difference between monaural and binaural audibility of speech is also greater, up to 6—9 db, when higher intensities are employed (Groen and Hellema 1960, Lochner and Burger 1961). As for the audibility of speech in noise, the same rules on phase difference hold good as for audibility of pure tones (Hirsh 1948 b, Licklider 1948, Feldman 1963).

In a study of the discrimination of speech it seems natural that, at least in normal undisturbed test conditions, the same discrimination score should be reached both monaurally and binaurally. Thanks to summation, 100 per cent intelligibility may be reached binaurally at a slightly lower intensity level. But in difficult listening condition — such as the »cocktail-party» situation — binaural discrimination is 10—20 per cent better (Jerger *et al.* 1961, Chappel *et al.* 1963). The most important contributory factor here is the decisively better localization of sound in binaural hearing. The ability to hear in noise is also definitely better binaurally than monaurally (Koenig 1950).

If speech signals presented to the two ears differ on points other than intensity, conditions change decisively. A situation in which completely different texts were presented to each ear was described above (p. 14). But if the same test words are divided into two by filtration and presented separately to the ears, the conditions produced are those of Matzker's test. The literature contains little information on the understanding of binaurally presented filtered speech. Fletcher (1929) reported on Arnold's findings concerning monaural and binaural hearing of speech divided into two at 1000 cps (see p. 15). Broadbent and Ladefoged (1957), using synthetic speech, found that if the first formant ($F_1$) of sounds was presented to one ear and the second formant ($F_2$) to the other, good intelligibility was ensured if both formants had the same fundamental frequency ($F_0$). Fusion failed to occur when the fundamental frequencies were different.

Matzker (1958) gave a short description of his observations concerning monaural and binaural discrimination with the frequency bands he used in his binaural test. With test subjects of normal hearing he found that the discrimination score for the band of 1500—2400 cps varied from 6 to 30 per cent and for the 500—800 cps band from 0 to 26 per cent when intensity was at the 30—40 db sensation level. When both bands were presented simultaneously to both ears the discrimination score could be raised to 97 per cent, and monaurally, both bands to the same ear, to 90 per cent. In the

binaural test conditions proper he considered 10 mistaken words out of 41 to be normal — a discrimination score of 75 per cent — and only when discrimination fell below 63 per cent (15 mistakes) was the result considered definitely pathological

When standardizing a binaural speech resynthesis test Lindén (1960, 1964) measured the discrimination scores for the 560—715 cps and 1800—2200 cps bands (one-third octave wide), separately for each band and also in combination both on a binaural and a monaural test The steepness of the attenuation of the filters he employed was very effective that of the low band 100 db per octave all the way to the 30 db level, though after this the attenuation fell rapidly, and that of the high band 200 db per octave, which remained steady up to 50 db The material Linden used in his test consisted of spondaic words specially selected for the binaural test Discrimination scores obtained for the low band, at sensation levels of 30, 40, 50 and 60 db, were 7, 14, 31 and 46 per cent, respectively The high band, with the same intensity values, gave 5, 11, 17 and 22 per cent discrimination On binaural presentation, discrimination scores for these bands, at sensation levels of 25, 30, 35 and 40 db, were 37, 64, 81 and 89 per cent On monaural and binaural presentation the test gave identical values

With a view to examinig a binaural hearing aid Huizing and Taselaar (1961), using PB words, studied discrimination scores for two octave-wide (140—280 cps and 1128—2256 cps) bands, both monaurally and binaurally There was no discrimination with the low band alone, whereas with the high band SRT (the speech reception threshold) was 34 db, and 100 per cent discrimination was reached at an intensity of 60 db When both bands were presented simultaneously, with the intensity of the low band constant at 76 db, SRT was 26 db in the monaural and only 12 db in the binaural testing conditions Binaural hearing in these conditions was thus better than monaural, with a difference in SRT of 14 db

No systematic studies of binaural discrimination with bands of varying widths taken from different parts of the speech area have been published The selection of the bands for Matzker's test was originally effected on the basis of selective tests, but the results of these tests have never been published The same is true of Linden's selection of bands It is obvious that in any attempt to apply the test concerned to various languages it is important to study the matter in detail in order to arrive at the best possible selection of bands and word material Since information on the discrimination of filtered speech is completely lacking in the Finnish language the following study has been undertaken to examine the influence of various filtration conditions on the intelligibility of Finnish speech

# III PURPOSE OF THE INVESTIGATION

The most important aim of the present investigation is to create a Finnish test, based on the use of filtered speech, for examination of central hearing disorders The following points are studied with normal test subjects, employing the vocabulary already in use in Finnish speech audiometry

— the effect of low pass and high pass filtration on the intelligibility of speech,

— the effect of different band widths, selected from different parts of the frequency area of speech, on speech discrimination, and

— the combination of different pairs of bands in a binaural study to find the bands best suited to a hearing synthesis test

These studies completed, word material suitable for hearing synthesis test will be selected Based on work with this material, and on earlier observations, a Finnish speech audiometric method for the diagnosis of central hearing disorders, intended for routine use, is assembled

# IV. EQUIPMENT AND MATERIAL

The principal equipment employed for the investigation is shown as a block diagram in Fig 7 In addition, a Beltone 15 A audiometer, calibrated according to the NBS standard, was used for the determination of the pure tone thresholds for each test person Test material was dictated on tape using a Labor MD 5 H microphone Its frequency response characteristics are shown in Fig 8

The tape recorder was of Grundig manufacture, and its frequency response characteristics can be seen in Fig 9

From the tape recorder the test words were transmitted to an amplifier of a special make, with the following values input 5 mV/5 MΩ and output 2 x 9 V/600Ω An insert type M 81 earphone intended for the examiner's use was connected to the amplifier

The most essential parts of the equipment were the filters Four Allison Model 2B filters were used, each comprising two separate filter

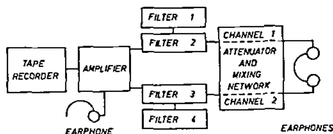
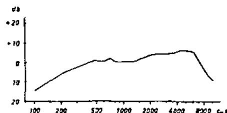



Fig 7 Block diagram of test equipment



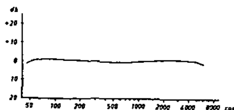




Fig 8 Frequency response characteristics of Labor MD 5 H microphone to a fixed input voltage (left)

Fig 9 Frequency response characteristics of Grundig tape-recorder to a fixed input voltage (right)

units, a low pass and a high pass filter The attenuation of one filter was 30 db per octave and by using two filters coupled in series it could be increased to 60 db The attenuation characteristics of the filters are given in Fig 10

After filtration the test material was fed into a Madsen Model OB 60 audiometer with two independent channels which could be combined if required This audiometer was calibrated according to the NPL standard

Monaural tests were made with Beltone TDH 39, and binaural tests with insert type M 81 earphones The characteristics of both pairs of earphones are shown in Fig 11

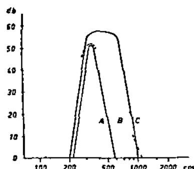



Fig 10

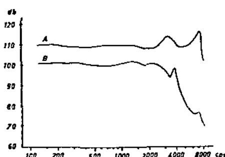



Fig 11

*Fig 10* Attenuation characteristics of Allison 2 B filters Curve A shows the attenuation of two filters connected in series with the narrow band Curve B shows the attenuation of one single filter with the narrow band, and Curve C the attenuation in an octave-wide band obtained by means of two filters coupled in series

*Fig 11* Frequency response characteristics of earphones Curve A shows the frequency response of Beltone TDH 39 earphones to a fixed 0.1 V input, measured by a 6 cc coupler Curve B shows the frequency response of insert-type M 81 earphones to a fixed 30 mV input, measured by a 2 cc coupler

The vocabulary for routine use in filtered speech tests was recorded after filtration with a two-channel Tandberg Model 72B tape recorder

The tests were carried out in the Audiologic Laboratory of the Oto-laryngologic Department of the University of Turku The test room was subject to very little ambient noise, particularly at night, when the present tests were effected Since the hearing of the test subjects was tested only through air conduction, the subjects' ears could be covered by earphones The influence of ambient noise on the speech tests effected at relatively high intensities was thus negligible

The investigation covered a total of 961 healthy test subjects of normal hearing ranging in age from 17 to 32 years The majority were young national servicemen, a number were medical students, nurses and other hospital staff None of them had any previous experience of listening to filtered speech and few knew anything about audiometric testing

TABLE 4

*Word lists I and II in Finnish speech audiometry* (T Palva 1952)

| List I | | List II | |
|---|---|---|---|
| jarvi | loppuu | isku | myyda |
| unto | torma | ehyt | lappi |
| *laskee* | *sangen* | *valo* | *keitos* |
| tuho | haiy | luonto | komea |
| ilma | mutta | varis | valjyys |
| kengan | silloin | kaden | nuori |
| tusina | noukku | lappu | ahti |
| jyra | julma | ehto | kuljen |
| leivo | yossa | syoda | rysa |
| osa | etta | kevat | tuuli |
| meni | nuoska | sulaa | seppa |
| törky | riepu | rumu | kova |
| potilas | mela | otti | muoti |
| yrjö | ysku | savel | sydan |
| keha | rungon | aamu | keula |
| puree | ylväs | kuppi | soutaa |
| möly | paukkuu | ylla | pelko |
| kaunis | miten | sangon | vaino |
| seppo | maksu | ruoste | rauta |
| virma | aly | läpi | iskee |
| löydan | pino | hiekka | oljy |
| vilkkuu | ujo | vuori | langon |
| matto | pare | pettaä | ruuti |
| saro | liukas | alku | köyha |
| arvo | suunnaton | soittaa | myohäan |
| lypsaa | kellui | luja | satoi |
| oikku | pyha | ehka | kiuru |
| peukalo | aalto | kaivaa | oja |
| jyva | kaytos | ohut | kylla |
| maistuu | meri | sääli | oisin |

Finnish speech audiometry lists No I and II (T Palva 1952) were used in the test as the basis of the phonetic material. These lists are phonetically balanced and intended for determination of auditory discrimination (Table 4) They were dictated onto magnetic tape at 5 second intervals by an experienced male speaker who simultaneously observed the oscilloscope of the tape recorder to keep the sound volume constant

# V. TECHNIQUE OF TESTING

First the pure tone threshold of each test subject was measured, by air conduction, at 125, 250, 500, 1000, 1500, 2000, 3000, 4000, 6000 and 8000 cps If the hearing at any of these frequencies was below 10 db, the test subject was not considered to have normal hearing and was not accepted for the speech test

All tests in connection with the present investigation were carried out by the author personally.

Before the speech test the test subject was told that at 5 second intervals he would hear through the earphones test words, all of which were familiar Finnish words though they might sound very unclear The test subject was asked to repeat the word as he understood it, as soon as he heard it The answers were immediately recorded A word was considered misunderstood if any of its sounds was incorrectly repeated

The word material was divided into 4 lists of 30 words by dividing each of the two original 60-word lists into two parts Each of the 4 word lists was delivered to the test subject with different frequency distortions so that he would certainly understand some words of one of the 30 word series In this way the subject's interest could more easily be kept alive At the same time care was taken to have each group of the words tested the same number of times at each frequency distortion

The cut-off values indicated by the instrument panel of the filters were *recorded as cut-off frequencies of filtration*

The technique for monaural tests differed slightly from that employed for binaural tests

## 1 MONAURAL TESTS

In order to reduce disturbing outside factors both ears were kept covered by earphones in monaural as in binaural tests In monaural tests TDH 39 earphones were used, since their reproduction characteristics, particularly in the high frequency area, were definitely better than those of the M 81 earphones With the latter the upper cut-off could not be raised above 3500 cps without the attenuation of the earphones themselves affecting the result

Since filtration clearly affects sound intensity when the energy input to the filter is constant, it was necessary to change intensities at different filter positions using the amplifier and the attenuator Preliminary tests by the

examiner revealed that at the 50 db sensation level an intensity was usually reached where hearing presented no more difficulty, and the fall in discrimination could be considered the outcome of filtration alone As a result of the attenuation characteristics of the filters, discrimination naturally still increased slightly when the intensity was raised, especially when narrow bands were used In particular, when high and narrow bands and the high cut-off values of high-pass filtration were employed the signal had to be amplified considerably (some 20—25 db), to reach the same sensation level as with the participation of low sounds or with the use of wide bands When the signal had to be heavily amplified, the background noise in the equipment also increased markedly It was, however, fairly slight in the majority of test conditions

Half the tests were carried out with the right ear and the other half with the left

## 2 BINAURAL TESTS

In binaural tests it is essential that overhearing from one ear to the other can be prevented For this reason it is necessary to use earphones providing the best possible interaural insulation and to keep testing intensity safely low The interaural insulation of the earphones used in the present tests was studied by Palva and Palva (1962) with pure tones and white noise The interaural insulation of TDH 39 earphones is 50—55 db, the insert-type M81 earphone gives results 15—20 db better For this reason all binaural tests in the present study were carried out using insert earphones

In order to assess the influence of interaural insulation in the binaural test conditions, experiments were conducted with five subjects who were deaf in one ear and had normal hearing in the other Tests were presented with the low band of 480—720 cps to the hearing ear at a constant 50 db sensation level the discrimination score never exceeded 45 per cent The deaf ear simultaneously received the high band (1560—2040 cps) beginning with the 60 db sensation level, intensity was then increased until discrimination improved clearly through overhearing The results (Fig 12) accord well with those with pure tones (Palva and Palva 1962) From these experiments it may be concluded that sensation levels exceeding 70 db cannot be applied in binaural tests on subjects with normal cochlear function

In all the binaural tests completed in connection with the present investigation an effort has been made to deliver the signals to both ears at the same subjective intensity The testing intensity chosen was a monaural sensation level of 50 db For each pair of bands, special intensity readings had to be determined with the aid of the amplifier and the attenuator, in order to reach the same sensation level The threshold of detectability was determined for each band to obtain a relative 0 db value, according to which the sensation level could be determined Furthermore, a subjective loudness comparison

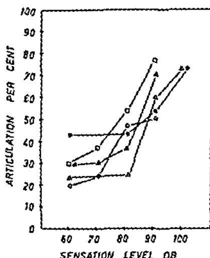



*Fig 12* Interaural insulation of insert-type M 81 earphones in binaural speech test conditions measured on five monaurally deaf patients The 480—720 cps band was given to the normal ear at a constant 50 db sensation level, and the 1560—2040 cps band to the deaf ear at the varying sound intensities indicated at the bottom of the diagram

was made prior to each test to ensure that the sensation levels of the band pairs employed on each occasion were identical

For half the number of those tested the high band was delivered to the right ear and the low band to the left, and for the other half vice versa

## 3 STATISTICAL TREATMENT

The statistical treatment of the present material was carried out by statistical routine methods in general use (e g Cramér 1945)

There have been observations of 5—20 subjects in each combination of test conditions The means and standard deviations of the observed discrimination scores were calculated for each of these points in the test series The resulting statistics describe the tests satisfactorily, especially where discrimination scores of 20 to 80 per cent are concerned The skewness of the distribution occurring with small and large values has not been taken into account in the calculations Since the tests are considered as a series this fact is of no essential significance in the study of the results

Comparison of two mean values or mean value groups have been made by applying the t-test, the difference has been termed significant when the corresponding value of the probability (P) has been $\leq 0\ 05$

For binaural tests a study was undertaken to see whether there were differences within the test groups between the different tests or whether any differences obtained in the test were due to chance Variance analysis of one-way classification was used for this purpose The situation studied was that in which one half of a pair of bands remained constant while the other was varied Differences were considered to exist between tests of a test series if $P \leq 0\ 05$

# VI RESULTS

## 1 MONAURAL TESTS

Initially, a series of tests on 20 subjects was carried out, using the equipment and technique described above, to present unfiltered the whole test material of 120 words The discrimination score obtained was 98 6 ± 1 9 per cent

### a) LOW PASS FILTRATION

With the technique described the frequency area was limited at its high end, using 15 cut off frequencies which ranged from 4080 cps to 450 cps The attenuation of the filters was 60 db per octave For this test series 57 test subjects were employed and each cut-off value of filtration was tested 15 times with lists of 30 words It was found that discrimination did not begin to decline until the cut-off frequency fell to about 2000 cps After this the drop was roughly rectilinear and reached the 35 per cent level at 600 cps Discrimination subsequently fell steeply to near zero at 570 cps, and at 450 cps only a few words could accidentally still be discerned

Between 600—1800 cps individual differences in discrimination were very great, at 780 cps for example, they ranged from 10 to 73 per cent When distortion was slight or very great the dispersal was markedly smaller The results are shown in Table 5 and Fig 13

The first mistakes to occur were evident in the discrimination of consonants At 2000 cps, typical mistakes were the addition of a consonant in front of a word beginning with a vowel, the substitution of *h* for *s*, and the confusion of *m*, *n* and *ŋ* Only after the cut-off frequency of filtration fell below 1500 cps did mistakes begin to occur in the discrimination of vowels

### b) HIGH PASS FILTRATION

The frequency area of speech was limited at the low end by using 14 cut-off frequencies ranging from 450 cps to 4080 cps Each degree of filtration was tested 15 times with lists of 30 words each using a total of 53 test subjects

Discrimination started to decline as soon as the cut-off frequency exceeded 600 cps and declined steadily to near zero at about 3000 cps Individual

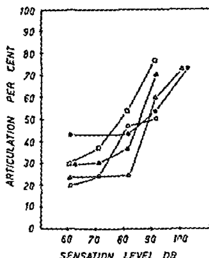



Fig 12 Interaural insulation of insert-type M 81 earphones in binaural speech test conditions measured on five monaurally deaf patients The 480—720 cps band was given to the normal ear at a constant 50 db sensation level and the 1560—2040 cps band to the deaf ear at the varying sound intensities indicated at the bottom of the diagram

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For binaural tests a study was undertaken to see whether there were differences within the test groups between the different tests or whether any differences obtained in the test were due to chance Variance analysis of one-way classification was used for this purpose The situation studied was that in which one half of a pair of bands remained constant while the other was varied Differences were considered to exist between tests of a test series if $P \leq 0\,05$

# VI RESULTS

## 1 MONAURAL TESTS

Initially, a series of tests on 20 subjects was carried out, using the equipment and technique described above, to present unfiltered the whole test material of 120 words The discrimination score obtained was 98 6 ± 1 9 per cent

### a) LOW PASS FILTRATION

With the technique described the frequency area was limited at its high end, using 15 cut off frequencies which ranged from 4080 cps to 450 cps The attenuation of the filters was 60 db per octave For this test series 57 test subjects were employed, and each cut-off value of filtration was tested 15 times with lists of 30 words It was found that discrimination did not begin to decline until the cut-off frequency fell to about 2000 cps After this the drop was roughly rectilinear and reached the 35 per cent level at 600 cps Discrimination subsequently fell steeply to near zero at 570 cps, and at 450 cps only a few words could accidentally still be discerned

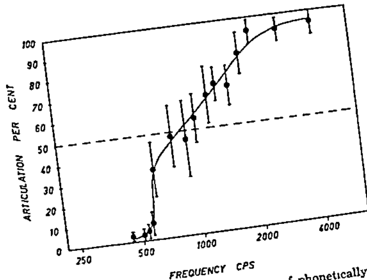
Between 600—1800 cps individual differences in discrimination were very great, at 780 cps, for example, they ranged from 10 to 73 per cent When distortion was slight or very great the dispersal was markedly smaller The results are shown in Table 5 and Fig 13

The first mistakes to occur were evident in the discrimination of consonants At 2000 cps typical mistakes were the addition of a consonant in front of a word beginning with a vowel, the substitution of *h* for *s* and the confusion of *m*, *n* and *ŋ* Only after the cut-off frequency of filtration fell below 1500 cps did mistakes begin to occur in the discrimination of vowels

### b) HIGH PASS FILTRATION

The frequency area of speech was limited at the low end by using 14 cut-off frequencies ranging from 450 cps to 4080 cps Each degree of filtration was tested 15 times with lists of 30 words each, using a total of 53 test subjects

Discrimination started to decline as soon as the cut-off frequency exceeded 600 cps and declined steadily to near zero at about 3000 cps Individual




ig 13 Effect of low-pass filtration on the intelligibility of phonetically balanced Finnish est words at optimal sound intensity Lines indicating the size of one standard deviation have been drawn, in this and in the following diagram, on both sides of the point indicating the mean value

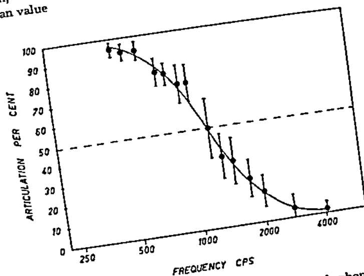



Fig 14 Effect of high-pass filtration on the intelligibility of phonetically balanced Finnish test words at optimal sound intensity

differences in discrimination were greatest when distortion was mediu strong (1020—1560 cps) The dispersal was, however, slighter than low-pass filtration, at 1200 cps, for instance, the extreme values were and 77 per cent The results are given in Table 6 and Fig 14

Mistakes in the discrimination of vowels were the first to appear more frequencies of the low end were eliminated more vowels were he as ı and y Consonant mistakes did not begin until the cut-off freque ex eeded 1300 cps

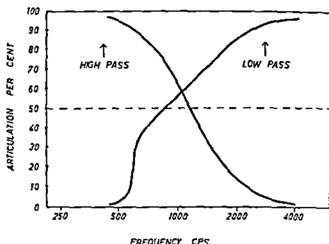



*Fig 15* Effect of low-pass and high pass filtration on the intelligibility of phonetically balanced Finnish test words at optimal sound intensity

In order to obtain a better idea of the influence of both the low-pass and the high-pass filtration, *especially in comparison* with investigations by other authors, the two curves were plotted in one and the same figure (Fig 15) The curves intersected at slightly above 1000 cps, where the discrimination score was about 60 per cent

c) BAND PASS FILTRATION

*Narrow band*

Narrow band in the present paper refers to a band obtained [illegible] cut-off values of both low pass and high pass filters are adjusted to [illegible] reading in the middle of the band

To start with, the changes in discrimination produced by [illegible] and one high pass filter were studied The attenuation of the [illegible] db/octave A total of 13 bands from 300 cps to 7200 cps, [illegible] and 30 word lists were used five times to test each band The [illegible] test subjects

Discrimination was found to be best at about 1200—2000 [illegible] 50 per cent for all test subjects, and was usually 70—90 [illegible] low frequency area at about 450—600 cps, discrimination [illegible] cent, then showed a declining trend at 700 cps but rose [illegible] frequency grew At the high end, discrimination was [illegible] level of over 50 per cent for some subjects even in the [illegible] bands while for the others the discrimination in these [illegible] zero The results of these tests are given in Table 7 [illegible]

TABLE 5

*Effect of low-pass filtration on discrimination scores*

| Cut-off frequency cps | 450 | 510 | 510 | 570 | 600 | 780 | 900 | 1020 | 1200 | 1320 | 1560 | 1800 | 2040 | 2880 | 4080 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mean per cent | 2 2 | 3 1 | 4 7 | 8 7 | 33 6 | 47 9 | 45 6 | 50 5 | 65 6 | 70 1 | 68 1 | 83 6 | 93 0 | 92 1 | 94 1 |
| S D | 2 4 | 3 2 | 3 5 | 5 9 | 13 1 | 15 0 | 17 8 | 12 0 | 13 8 | 8 0 | 9 3 | 10 1 | 6 0 | 5 9 | 6 0 |

TABLE 6

*Effect of high-pass filtration on discrimination scores*

| Cut-off frequency cps | 450 | 510 | 600 | 720 | 780 | 900 | 1020 | 1200 | 1320 | 1560 | 1800 | 2040 | 2880 | 4080 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mean per cent | 94 6 | 92 1 | 92 3 | 81 0 | 79 3 | 73.9 | 74 0 | 49 7 | 34 4 | 32 4 | 22 6 | 13 9 | 4 5 | 2 2 |
| S D | 4 2 | 4.8 | 5 7 | 7 4 | 5 9 | 10 2 | 9 4 | 15 2 | 12 6 | 12 4 | 8 2 | 8 2 | 6.2 | 3 7 |

TABLE 7

*Discrimination scores with narrow bands in different frequency areas*
*Filter attenuation 30 db/octave*

| Cut-off frequency cps | 300 | 360 | 450 | 510 | 600 | 720 | 900 | 1200 | 1560 | 2040 | 2880 | 4800 | 7200 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mean per cent | 0 | 6 | 29 | 21 | 22 | 18 | 47 | 66 | 81 | 60 | 50 | 29 | 30 |
| S D | 0 | 6 | 11 | 6 | 11 | 11 | 25 | 5 | 12 | 11 | 16 | 27 | 26 |

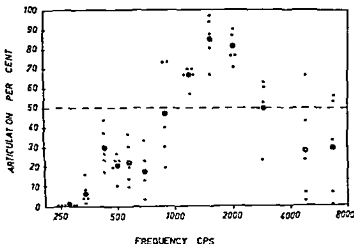



Fig 16 Intelligibility of narrow bands in different frequency areas Filter attenuation is 30 db/octave and testing intensity at a sensation level of 50 db In this and the following diagrams the small dots indicate individual and the large dots mean values

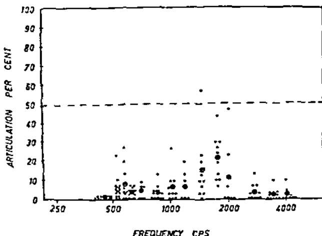



Fig 17 Intelligibility of narrow bands in different frequency areas Filter attenuation is 60 db/octave and testing intensity at a sensation level of 50 db The same filter attenuation and sensation level is used in Figs. 18—38

information obtained on even this small series of subjects was considered enough to show that this filter attenuation was not adequate for a hearing synthesis test

The next step was to study narrow bands with a filter attenuation of 60 db octave Two low-pass and two high-pass filters were employed in the test, coupled in series Discrimination over a total of 14 bands, from 450 cps to 4080 cps, was analysed The intelligibility of each band was determined ten times with 30-word lists, and 35 persons were tested In these conditions discrimination was very poor in all bands, only accidentally did it rise to

TABLE 8

*Discrimination scores with narrow bands in different frequency areas*
*Filter attenuation 60 db/octave*

| Cut off frequency cps | 450 | 540 | 570 | 600 | 720 | 900 | 1020 | 1200 | 1560 | 1800 | 2040 | 2880 | 3600 | 4080 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mean per cent | 0.7 | 5.3 | 8.3 | 3.3 | 4.0 | 3.0 | 7.3 | 6.3 | 15.0 | 21.3 | 11.3 | 3.3 | 1.7 | 1.3 |
| SD | 2.0 | 7.2 | 9.0 | 2.7 | 3.5 | 4.3 | 9.0 | 6.6 | 16.4 | 11.5 | 14.7 | 4.4 | 3.3 | 3.1 |

TABLE 9

*Discrimination scores with 1/4 octave wide bands in different frequency areas*

| Mean frequency cps | 330 | 405 | 480 | 540 | 570 | 600 | 660 | 810 | 1020 | 1200 | 1320 | 1740 | 2040 | 2400 | 2640 | 3240 | 3720 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Low cut-off cps | 300 | 360 | 420 | 480 | 510 | 540 | 600 | 720 | 900 | 1080 | 1200 | 1560 | 1800 | 2160 | 2400 | 2880 | 3360 |
| High cut-off cps | 360 | 450 | 540 | 600 | 630 | 660 | 720 | 900 | 1140 | 1320 | 1440 | 1920 | 2280 | 2640 | 2880 | 3600 | 4080 |
| Mean per cent | 0 | 1.7 | 3.0 | 13.7 | 11.0 | 18.0 | 8.7 | 7.7 | 7.3 | 22.3 | 19.7 | 22.3 | 21.0 | 4.0 | 8.7 | 5.0 | 5.3 |
| SD | 0 | 2.5 | 3.6 | 7.5 | 6.8 | 6.6 | 4.4 | 5.9 | 5.7 | 14.3 | 15.0 | 22.8 | 9.2 | 6.5 | 11.6 | 5.2 | 5.0 |

TABLE 10

*Discrimination scores with 1/3 octave wide bands in different frequency areas*

| *Mean frequency cps* | 285 | 345 | 420 | 495 | 570 | 585 | 630 | 690 | 810 | 1050 | 1260 | 1380 | 1800 | 2100 | 2520 | 2760 | 3360 | 3840 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Low cut-off cps | 240 | 300 | 360 | 420 | 480 | 510 | 540 | 600 | 720 | 900 | 1080 | 1200 | 1560 | 1800 | 2160 | 2400 | 2880 | 3360 |
| High cut-off cps | 330 | 390 | 480 | 570 | 660 | 660 | 720 | 780 | 960 | 1200 | 1440 | 1560 | 2040 | 2400 | 2880 | 3120 | 3840 | 4320 |
| Mean per cent | 1.3 | 1.0 | 1.7 | 3.3 | 15.0 | 14.3 | 22.7 | 5.7 | 8.7 | 18.0 | 29.3 | 34.7 | 21.3 | 15.3 | 11.0 | 8.0 | 5.0 | 2.7 |
| SD | 3.1 | 1.6 | 2.9 | 5.0 | 6.2 | 6.7 | 14.0 | 4.7 | 8.0 | 9.3 | 14.7 | 19.5 | 15.6 | 12.3 | 10.2 | 9.7 | 6.0 | 5.0 |

roughly 50 per cent at about 1560—2040 cps The best discrimination was obtained with bands 1560 cps (15 0 ± 16 4 per cent), 1800 cps (21 3 ± 11 5 per cent) and 2040 cps (11 3 ± 14 7 per cent) At the low end some distinctly better discrimination scores were recorded for 540 and 570 cps bands than for 700 and 900 cps bands The results of this series of tests are given in Table 8 and Fig 17

*1/4 octave band*

The filter attenuation used in this and all the following tests was 60 db per octave The bands studied covered the frequency area from 300 cps to 4080 cps 43 test subjects were employed for the study of 17 bands, and each band was tested ten times with 30-word lists

Discrimination was found to remain usually below 30 per cent and to rise only accidentally to around 50 per cent at about 1300—1700 cps At the low end, below a mean frequency (arithmetic mean of the cut-off frequencies) of 500 cps no test subject reached a discrimination score exceeding 10 per cent, while in the range 540—600 cps average discrimination amounted to as much as almost 20 per cent (18 0 ± 6 6 per cent in the band of 540—660 cps) Approaching 1000 cps discrimination was again poorer (in the band 900—1140 cps only 7 3 ± 5 7 per cent) and it reached its maximum in the bands 1080—1320 cps, 1200—1440 cps, 1560—1920 cps and 1800—2280 cps, in which it was of the same order, about 20 per cent When the mean frequency exceeded 2000 cps discrimination scores fell rapidly almost to zero For all bands there were subjects whose discrim nation scores were roughly zero The results of this series of tests are given in Table 9 and Fig 18

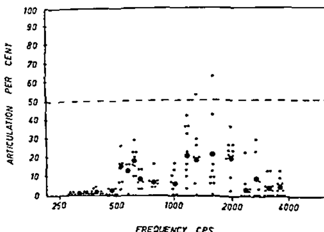



Fig 18 Intelligibility of 1/4 octave wide bands in different frequency areas The dots are placed at the arithmetic means of the cut-off frequencies, similarly to Figs. 19—22.

*1/3 octave band*

The frequency area studied covered a total of 18 bands between 240 cps and 4320 cps Each band was tested ten times with 30-word lists The number of test persons was 45 Two discrimination peaks could be discerned, a lower at about 600 cps (band 540—720 cps, 22 7 ± 14 0 per cent) and a higher at about 1400 cps (band 1200—1560 cps, 34 7 ± 19 5 per cent) Again, a few test persons had discrimination scores approaching zero in all the bands studied. The results are given in Table 10 and Fig 19

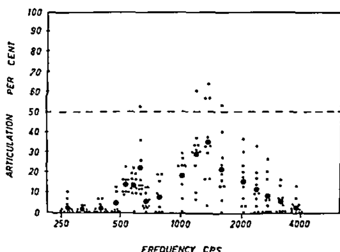



Fig 19 Intelligibility of 1/3 octave wide bands in different frequency areas

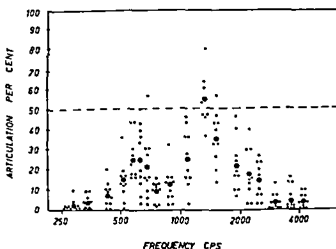



Fig 20 Intelligibility of 1/2 octave wide bands in different frequency areas

*1/2 octave band*

The frequency area studied covered a total of 18 bands between 240 cps and 5040 cps Each band was tested ten times with 30-word lists 45 test persons were used Again, there were two discrimination peaks visible, one at about 600—700 cps (band 480—720 cps, 25 7 ± 10 4 per cent, band 510—780 cps, 26 0 ± 15 0 per cent, and band 540—840 cps, 21 0 ± 15 5 per cent) and the other at about 1400 cps (band 1080—1680 cps, 54 1 ± 11.8 per cent). For the band with the best discrimination score none of the test persons showed scores below 30 per cent The results are given in Table 11 and Fig 20

*One-octave band*

The frequency area studied covered a total of 18 bands between 240 cps and 6720 cps 45 test subjects were used and each band was tested ten times with 30-word lists Maximum discrimination was found in bands 900—1800 cps (52 0 ± 12 6 per cent) and 1080—2160 cps (54 0 ± 12 7 per cent) There were no essential differences in discrimination when the mean frequencies ranged from 600 to 1000 cps, the scores remaining low, at about 25—30 per cent In the most important speech frequency area (mean frequencies between 500 and 2000 cps) scores of 10 per cent or below were accidental The results are given in Table 12 and Fig 21.

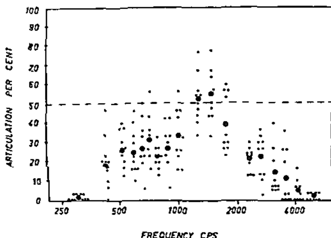



Fig 21 Intelligibility of 1 octave wide bands in different frequency areas

*1 ½ octave band*

The frequency area studied ranged from 240 to 7200 cps, and a total of ten bands were used. Test subjects numbered 25, and each band was tested

TABLE 11

*Discrimination scores with 1/2 octave wide bands in different frequency areas*

| Mean frequency cps | 300 | 375 | 450 | 525 | 600 | 645 | 690 | 750 | 900 | 1110 | 1380 | 1500 | 1980 | 2220 | 2640 | 3000 | 3600 | 4200 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Low cut-off cps | 240 | 300 | 360 | 420 | 480 | 510 | 540 | 600 | 720 | 900 | 1080 | 1200 | 1560 | 1800 | 2160 | 2400 | 2880 | 3360 |
| High cut-off cps | 360 | 450 | 540 | 630 | 720 | 780 | 840 | 900 | 1080 | 1320 | 1680 | 1800 | 2400 | 2640 | 3120 | 3600 | 4320 | 5040 |
| Mean per cent | 1.3 | 3.3 | 7.7 | 14.3 | 25.7 | 26.0 | 21.0 | 9.7 | 12.3 | 26.0 | 54.0 | 35.7 | 22.3 | 18.0 | 14.0 | 3.7 | 4.7 | 4.3 |
| SD | 3.1 | 4.2 | 6.3 | 9.9 | 10.4 | 15.0 | 15.5 | 4.8 | 9.9 | 14.3 | 11.8 | 13.0 | 13.1 | 11.9 | 9.0 | 4.3 | 6.3 | 3.8 |

TABLE 12

*Discrimination scores with 1 octave wide bands in different frequency areas*

| lean irequency cps | 360 | 450 | 540 | 630 | 720 | 765 | 810 | 900 | 1080 | 1350 | 1620 | 1800 | 2340 | 2700 | 3240 | 3600 | 4320 | 5040 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| cut-off cps | 240 | 300 | 360 | 420 | 480 | 510 | 540 | 600 | 720 | 900 | 1080 | 1200 | 1560 | 1800 | 2160 | 2400 | 2880 | 3360 |
| ch cut-off cps | 480 | 600 | 720 | 840 | 960 | 1020 | 1080 | 1200 | 1440 | 1800 | 2160 | 2400 | 3120 | 3600 | 4320 | 4800 | 5760 | 6720 |
| per cent | 1.3 | 19.3 | 26.3 | 24.3 | 27.7 | 32.3 | 23.3 | 28.0 | 33.3 | 52.0 | 54.0 | 39.7 | 21.0 | 22.3 | 13.7 | 11.7 | 4.7 | 2.3 |
| | 1.6 | 13.0 | 9.2 | 12.6 | 4.5 | 11.2 | 6.4 | 11.1 | 14.8 | 12.6 | 12.7 | 15.0 | 5.9 | 11.3 | 11.6 | 14.3 | 5.4 | 3.7 |

ten times with 30-word lists. Discrimination scores were now remarkably high, for all test subjects they amounted to 50 per cent or more in bands 510—1560, 600—1800, 720—2160 and 900—2640 cps, in which the average scores approximated 75 per cent. The results are given in Table 13 and Fig. 22.

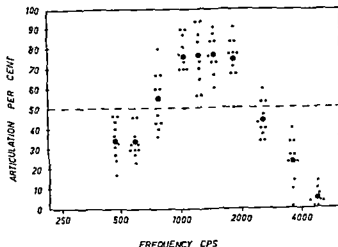



*Fig. 22.* Intelligibility of 1 1/2 octave wide bands in different frequency areas.

TABLE 13

*Discrimination scores with 1 ½ octave wide bands in different frequency areas*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Mean frequency cps | 480 | 600 | 810 | 1035 | 1200 | 1440 | 1770 | 1400 | 3540 | 4800 |
| Low cut-off cps | 240 | 300 | 420 | 510 | 600 | 720 | 900 | 1200 | 1800 | 2400 |
| High cut-off cps | 720 | 900 | 1200 | 1560 | 1800 | 2160 | 2640 | 3600 | 5280 | 7200 |
| Mean per cent | 34.3 | 34.0 | 54.3 | 75.3 | 75.7 | 76.0 | 74.3 | 44.0 | 23.0 | 5.0 |
| S.D. | 10.2 | 7.7 | 14.3 | 12.1 | 8.9 | 9.2 | 11.3 | 9.2 | 12.4 | 4.2 |

## *Relative importance of the different frequency areas for the discrimination of band-pass filtered speech*

Results from the tests described above relating to bands of varying widths were combined to obtain a better idea of the part played by the different frequency areas in the understanding of band-pass filtered speech. The results of the following tests were taken: the narrow band (attenuation 60 db/octave), 1/4, 1/3, 1/2 and one-octave band. On combination, an attempt was made to give each of the tests equal importance. The arithmetic mean frequency was chosen to represent each band. Since the mean frequencies of bands of varying widths differed slightly, the frequency area studied was

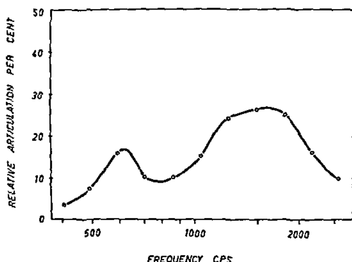



Fig 23 Intelligibility of band-pass filtered speech at different points of the most important frequency area of speech (The curve was obtained by combining the results illustrated in Figs 17—21)

divided into parts at 1/5 octave intervals (417 cps, 500 cps, 600 cps, and so on up to 3094 cps) The mean value curves of all five component tests were plotted into the same figure, and observation points were determined for each curve at 1/5 octave intervals by rectilinear interpolation

In order that each component test should carry the same weight, the reading of each observation point was reduced to higher terms in the following way, the sums of the values of the different observation points of each component test were made identical, by which means 5 curves of nearly the same arbitrary level were produced By drawing the mean value curve of these five curves the slightly idealized result in Fig 23 was obtained representing the intelligibility of band-pass filtered speech in different parts of the frequency area

## 2 BINAURAL TESTS

*Narrow bands*

The tests made with only a few subjects revealed that, if narrow bands were used, discrimination could be raised above 50 per cent only accidentally, even if the most favourable bands were selected (570 cps and 1800 cps) Further work with this arrangement was therefore discontinued

*1 4-octave bands*

A methodical search for bands suitable for the binaural test started among the bands 1 4 octave wide The low bands employed in the test were

420—540 cps, 480—600 cps, 540—660 cps, 600—720 cps and 720—900 cps and the high bands 1200—1440 cps 1560—1920 cps, 1800—2280 cps. 2160—2640 cps and 2400—2880 cps

All the five low bands listed above were tested with each of the listed high bands delivered in turn to the contralateral ear In this test series therefore, 25 different band combinations were tested presenting 30-word lists ten times each. A total of 63 test subjects was examined in this test. The results are given in Table 14 and Figs. 24—28

When the low band used was 420—540 cps average discrimination remained poor, the maximum being 31.7 ± 13 6 per cent (with the band 1560—1920 cps) The poorest pairs with this low band were those with 2160—2640 cps (9 0 ± 7 7 per cent) and 2400—2880 cps (10.3 ± 10.5 per cent) Only in two cases did discrimination exceed 50 per cent.

When the low band was 480—600 cps discrimination in almost all tests rose above the 50 per cent level. The highest mean discrimination was again obtained in combination with the band 1560—1920 cps (59.3 ± 13 1 per cent) even though the difference between this and discrimination with bands 1200—1440 cps (56 0 ± 16 6 per cent) and 1800—2280 cps (57 7 ± 16 6 per cent) was not significant. Discrimination with the band 2400—2880 cps (49 0 ± 14.3 per cent) was also of nearly the same order, whereas the band 2160—2640 cps gave a lower discrimination score (38.3 ± 20 0 per cent)

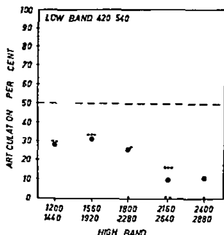
When the low band was 540—660 cps the results obtained were roughly similar to those of the preceding test. Mean discrimination was again best with the band 1560—1920 cps (61.3 ± 11.3 per cent) and poorest with 2160—2640 cps (44.7 ± 12.6 per cent)

When the low band was raised to 600—720 cps mean discrimination compared with the preceding test was slightly reduced. Results in the region of 50 per cent were obtained with the bands 1560—1920 cps, 1800—2280 cps and 2400—2880 cps (46.3 ± 14 7 per cent 52.3 ± 13 7 per cent and 41 7 ± 18 7 per cent respectively)

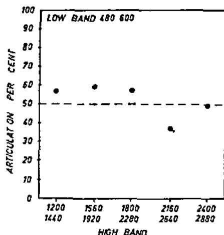
When the low band was 720—900 cps mean discrimination scores deteriorated still further and were near the 30 per cent level with all the high bands The individual values showed great variation e g with the band 1560—1920 cps from 3 to 70 per cent.

*1 3 octave bands*

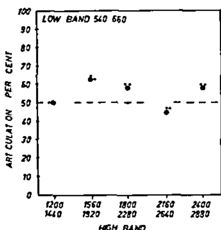
The tests with 1 3 octave bands were carried out in identical fashion to the series described above for the 1 4 octave wide bands. The low bands employed were 420—570 cps 480—660 cps, 540—720 cps 600—780 cps and 720—960 cps and the high bands 1200—1560 cps 1560—2040 cps, 1800—2400 cps 2160—2880 cps and 2400—3120 cps. The results of the tests carried out on 63 test subjects are given in Table 15 and Figs. 29—33



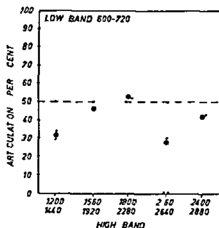

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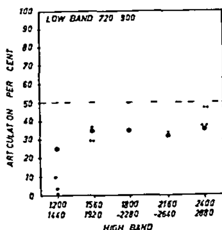

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*Figs 24—28 Intelligibility of two 1/4 octave wide bands* Here and in the tests illustrated in Figs 29—38 both bands were presented simultaneously to different ears at a 50 db monaural sensation level The low band presented to one ear is entered in the upper part of each diagram and the high band presented to the other ear at the bottom

TABLE 14

*Mean discrimination scores and standard deviations with pairs of 1/4 octave wide bands*

| | | High band cps | | | | |
|---|---|---|---|---|---|---|
| | | 1200—1440 | 1560—1920 | 1800—2280 | 2160—2640 | 2400—2880 |
| Low band cps | 420—540 | 28.0 + 7.7 | 31.7 ± 13.6 | 26.7 ± 16.3 | 9.0 ± 7.7 | 10.3 ± 10.5 |
| | 480—600 | 56.0 ± 16.6 | 59.3 ± 13.1 | 57.7 ± 16.6 | 38.3 ± 20.0 | 49.0 ± 14.3 |
| | 540—660 | 51.0 + 13.1 | 61.3 ± 11.3 | 58.3 ± 14.3 | 44.7 ± 12.6 | 59.0 ± 10.1 |
| | 600—720 | 31.3 ± 13.6 | 46.3 ± 14.7 | 52.3 ± 13.7 | 28.0 ± 18.4 | 41.7 ± 18.7 |
| | 720—900 | 25.3 ± 19.7 | 34.3 ± 21.4 | 34.0 ± 19.5 | 31.0 ± 9.7 | 34.0 ± 17.6 |

TABLE 15

*Mean discrimination scores and standard deviations with pairs of 1/3 octave wide bands*

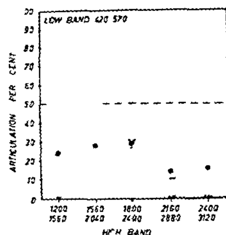
| | | High band cps | | | | |
|---|---|---|---|---|---|---|
| | | 1200—1560 | 1560—2040 | 1800—2400 | 2160—2880 | 2400—3120 |
| Low band cps | 420—570 | 23.3 ± 14.6 | 28.0 ± 13.4 | 28.3 ± 17.5 | 13.7 ± 16.7 | 16.0 ± 17.6 |
| | 480—660 | 46.0 ± 15.3 | 57.3 ± 11.6 | 61.0 ± 13.0 | 44.7 ± 18.7 | 46.0 ± 20.7 |
| | 540—720 | 43.0 ± 20.4 | 55.7 ± 14.1 | 54.7 ± 12.9 | 47.3 ± 19.4 | 45.7 ± 15.3 |
| | 600—780 | 25.0 ± 9.3 | 44.7 ± 13.5 | 47.3 ± 19.7 | 28.7 ± 20.5 | 26.3 ± 19.1 |
| | 720—960 | 23.3 ± 11.1 | 40.0 ± 17.8 | 41.0 ± 13.4 | 26.0 ± 11.5 | 29.7 ± 17.9 |

When the low band was 420—570 cps the mean values of discrimination were found to remain below 30 per cent, maximum discrimination being obtained in combination with the band 1800—2400 cps (28.3 ± 17.5 per cent). The same readings were also achieved when the high band was 1560—2040 cps (28.0 ± 13.4 per cent) or 1200—1560 cps (23.3 ± 14.6 per cent), whereas the scores obtained with higher bands were lower with 2160—2880 cps 13.7 ± 16.7 per cent and with 2400—3120 cps 16.0 ± 17.6 per cent.

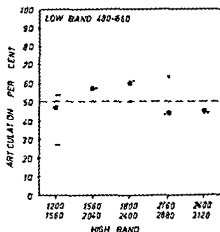
When the low band was 480—660 cps the mean discrimination scores with all the high bands tested rose to the neighbourhood of 50 per cent. Again, maximum scores were obtained with the bands 1560—2040 cps and 1800—2400 cps (57.3 ± 11.6 per cent and 61.0 ± 13.0 per cent).

When the low band was raised to 540—720 cps the results given in the preceding paragraph did not change appreciably, and only the bands 1560—2040 cps and 1800—2400 cps gave mean values of discrimination exceeding 50 per cent (55.7 ± 14.1 per cent and 64.7 ± 12.9 per cent).

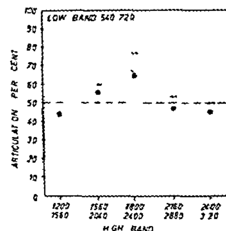
When the low band of 600—780 cps was used the discrimination fell slightly as compared with the preceeding two test groups, and the mean value remained below 50 per cent throughout all pairs of bands. Combination with the same pairs of bands as earlier yielded the best results 1560—2040



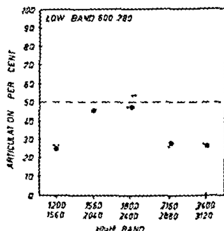

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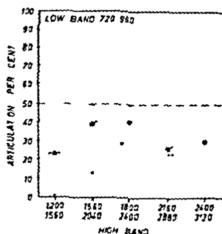

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32




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Figs 29—33 Intelligibility of two 1/3 octave wide bands

cps, 44 7 ± 13 5 per cent and 1800—2400 cps, 47 3 ± 19 7 per cent With the remaining three pairs of bands the discrimination score was about 30 per cent

With a low band of 720—960 cps the best mean values of discrimination were somewhere about 40 per cent (1560—2040 cps 40 0 ± 17 8 per cent and 1800—2400 cps 41 0 ± 13 4 per cent)

*Low band 1/2 octave and high band 1 3 octave*

The third series of binaural tests was carried out using the low bands of 420—630 cps, 480—720 cps, 540—840 cps, 600—900 cps and 720—1080 cps, and the high bands of 1200—1560 cps, 1560—2040 cps, 1800—2400 cps, 2160—2880 cps and 2400—3120 cps This series of tests was identical in technique with the previous series, though twice as many tests as in the earlier series were carried out with the six pairs of bands that seemed best The total number of test subjects in this series was 78 The results are given in Table 16 and Figs 34—38

With the low band of 420—630 cps the mean discrimination scores recorded with the most favourable high bands approached 50 per cent with 1200—1560 cps, 45 7 ± 15 5 per cent, with 1560—2040 cps, 44 7 ± 11 3 per cent, and with 1800—2400 cps, 49 3 ± 13 7 per cent The values obtained with the two highest bands, however, were poorer with 2160—2880 cps 31 7 ± 13 7 per cent and with 2400—3120 cps, 32 7 ± 13 4 per cent

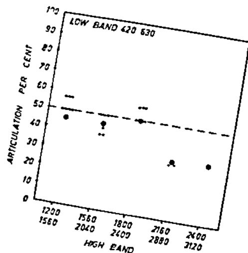
When the low band was raised to 480—720 cps, the mean value of discrimination rose to the neighbourhood of 70 per cent with the best pair of bands with the high band of 1560—2040 cps the discrimination score was 69 2 ± 12 8 per cent The results obtained with the bands 1800—2400 cps and 2160—2880 cps were of the same order (63 2 ± 17 5 per cent and 63 3 ± 21 3 per cent) With the bands 1200—1560 cps and 2400—3120 cps the results remained poorer (49 3 ± 12 5 per cent and 52 0 ± 25 5 per cent)

The low band of 540—840 cps gave the best mean value for discrimination together with the band 1560—2040 cps 70 5 ± 13 2 per cent The result was

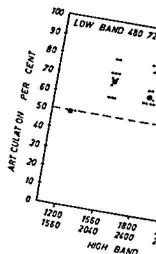
TABLE 16

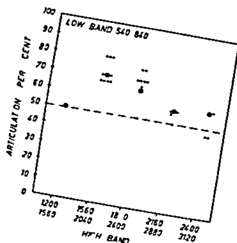
*Mean discrimination scores and standard deviations with pairs of 1/2 octave wide low and 1/3 octave wide high bands*

| | High band cps | | | | |
|---|---|---|---|---|---|
| Low band cps | 1200—1560 | 1560—2040 | 1800—2400 | 2160—2880 | 2400—3120 |
| 420— 630 | 45 7 ± 15.5 | 44 7 ± 11.3 | 49.3 ± 13 7 | 31 7 ± 13 7 | 32 7 ± 13 4 |
| 480— 720 | 49.3 + 12.5 | 69.2 ± 12.8 | 63.2 ± 17.5 | 63.3 ± 21.3 | 52 0 ± 25.5 |
| 540— 840 | 51 0 ± 17 6 | 70.5 ± 13.2 | 64.8 ± 15.3 | 57 7 ± 15 6 | 59.3 ± 13.3 |
| 600— 900 | 32.3 ± 7 0 | 48 7 ± 17.8 | 41.0 ± 15 1 | 34 7 ± 18 1 | 39 0 ± 18 4 |
| 720—1080 | 24 7 ± 16.5 | 35 7 ± 22.1 | 43 0 ± 14.8 | 31 7 ± 15 4 | 33 0 ± 21.5 |

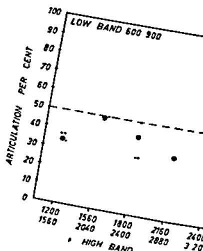


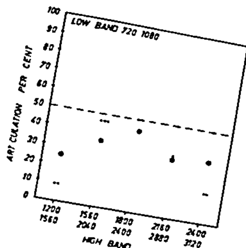

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34—38 Intelligibility of 1 2 octave wide low and 1/3 octave high bands in pairs

almost as good with the high band 1800—2400 cps (64 8 ± 15 3 per cent) With the 1200—1560 cps, 2160—2880 cps and 2400—3120 cps band the mean discrimination scores were 51 0 ± 17 6 per cent, 57 7 ± 15 6 per cent and 59 3 ± 13 3 per cent respectively

Raising the low band to 600—900 cps led to a deterioration in discrimination from the levels of the two preceding tests All mean discrimination scores were below 50 per cent, the highest was 48 7 ± 17 8 per cent, with the band 1560—2040 cps With the other bands (1200—1560 cps, 1800—2400 cps, 2160—2880 cps and 2400—3120 cps) the discrimination scores were poorer (32.3 ± 7 0 per cent, 41 0 ± 15 1 per cent, 34 7 ± 18 1 per cent, and 39 0 ± 18 4 per cent respectively)

When 720—1080 cps was used as the low band the mean discrimination scores were with 1200—1560 cps, 24 7 ± 16 5 per cent, with 1560—2040 cps 35 7 ± 22 1 per cent, with 1800—2400 cps, 43 0 ± 14 8 per cent, with 2160—2880 cps, 31 7 ± 15 4 per cent and with 2400—3120 cps 33 0 ± 21 5 per cent

# VII. DISCUSSION

*Any discussion of the intelligibility of distorted speech* (in this case by filtration) must take into account a few factors common to all such tests and affecting their results Individuals' ability to understand distorted speech differs considerably, even when all have had equal experience of tests of this kind (Licklider and Miller 1951, Owens 1961). One contributory factor *is, for example, a knowledge of the statistics of the language* which, especially with meaningful word material, plays a very important part in discrimination With such knowledge it is possible to fill in by guesswork even major gaps in the signal heard and to form an intelligible word or sentence (Fry 1964) Meaningless syllables as test material reduce this *possibility to a minimum, and the pure influence of distortion on the signal* presented emerges at its best

Since the ultimate aim of the present investigation is to produce a test by which information on central hearing ability, and in particular on the understanding of speech, can be obtained, meaningful words were chosen as *test material In the understanding of speech many central occurrences*, primarily of an associative nature are added to pure perception Sentences are considered the most reliable test material for the investigation of central functions (Bocca and Calearo 1963) The use of sentences, however, circumscribes the test to some extent, since making the test sufficiently *complex also makes it very long and tiring Much the same information* as that achieved from sentences is obtainable by means of spondee-type words (Fry 1964), which have proved the most serviceable in the filtered speech test (Linden 1960) Since in Finnish the emphasis is always on the first syllable of the word the corresponding Finnish word material is *trochaic All the tests carried out for the present study have for the above* reasons been performed with words of this type

For the study of the influence of filtration on the intelligibility of speech in Finnish two phonetically balanced lists of test words were employed In this way some idea of discrimination was obtained for the Finnish language as a whole This principle was abandoned only when a start was made on developing the material required for the filtered speech test, in an attempt to find the word material which best met the special requirements of the test

The selection of the test subjects plays a decisive part in discrimination tests Since the present investigation is aimed at developing a test for routine clinical use, the test subjects were chosen so as to represent as closely as possible the patient material to be examined in the future, they

should thus be representative of a very heterogeneous group To prevent the influence of degenerative changes on the results, however, only young people were considered eligible Almost all test subjects were, moreover, completely unaccustomed to listening to distorted speech and to audiometric examinations in general. Since training definitely improves a test subject's ability to understand distorted speech (Egan 1948, Licklider and Miller 1948), it is obvious that much higher discrimination scores could have been achieved using similar test material but trained test subjects, as was done by *e g* Hirsh *et al* (1954) The results reported in the present study are, because of this selection of test subjects, characterized by a very great dispersion, which makes final detailed conclusions somewhat difficult

## 1 MONAURAL TESTS

### a) LOW-PASS AND HIGH PASS FILTRATION

A study of the curves in Figs 13, 14 and 15 reveals that the low-pass and high-pass curves are different in shape Since most speech power in the low frequency area is in and around the fundamental frequency and the first vowel formant, it seems natural that this fact should emerge in these tests A study of the low-pass filtration curve shows that the hearing of fundamental frequency alone naturally does not make possible any discrimination of speech Only after the first formants of the vowels enter at about 600 cps is there a sudden rise in intelligibility, and the rise continues slowly but steadily while the second formants of different sounds become audible With cut-off frequencies below 1500 cps discrimination is based mainly on an identification of the vowels while consonant discrimination is more haphazard Above the 1500 cps level, as consonant mistakes diminish, discrimination increases fairly fast to its maximum

In high-pass filtration the conditions are rather different A study of the high pass curve reveals that elimination of fundamental frequency and of the first vowel formant does not appreciably affect discrimination Only when the cut-off frequency rises to the neighbourhood of 1000 cps does a very distinct drop occur in discrimination One might conclude that the majority of vowels can be identified without the first formant It must be borne in mind, however, that as a result of filter attenuation, the signal still includes at the cut-off frequency of 1000 cps sounds as low as 700 cps, though lower in intensity Once filtration begins to extend to the area of the second formant, discrimination falls distinctly as the formants in question are eliminated It might therefore be expected that discrimination should already fall to near zero at about 3000 cps when, taking filter attenuation into account, even the second formants of many vowels have been eliminated by filtration. This is in fact evident from the high-pass curve The very steady decline of the curve is obviously due to the dispersion of the second

vowel formant over a relatively wide frequency area (600—2500 cps) The words containing vowels discriminable from mere high tones such as i and e, are still discernible at the cut-off frequency of 2000 cps One reason for this is that the identification of consonants is based mainly on high tones, and mistakes in the discrimination of consonants do not occur until the cut off frequency is beyond 1300 cps A difference similar to that described above between the low-pass and high-pass filtration curves is also evident in the investigations made by Pollack (1948 b), and especially in those made by Miller and Nicely (1955)

Compared with most other languages, Finnish is very rich in vowels (Hakulinen 1941) On this basis a difference in filtration curves, compared for example with English, is to be expected since low tones would be more important for discrimination in Finnish The low-pass filtration curve does show a marked rise in discrimination with the entry of the first formant The subsequent course of the curve is more horizontal than in most English language studies (French and Steinberg 1947, Pollack 1948 b, Owens 1961) Similarly, concerning the high-pass curve, it is found that when tones important in the discrimination of vowels are eliminated, there is a drop in discrimination of Finnish material at relatively low cut-off values These factors tend to push both curves towards the low end of the frequency area, as can easily be seen from the location of the area where the curves intersect

There are other contributory factors to the location of the intersection of the present curves One of them is of an almost purely technical nature In tests in which the high cut-off values (above 2000 cps) of high pass filtration were used, sound intensity had to be amplified considerably (about 20—25 db) in order to obtain the same loudness as when low frequencies were included At the same time, however, the background noise caused by the equipment, which was otherwise negligible, became stronger Even though the speech to noise ratio remained unchanged, reinforced noise masks high tones more than it does low tones (Miller 1951) with the result that discrimination falls more rapidly In the same conditions, however, there is another factor of opposite effect with high cut-off values of high-pass filtration discrimination, with increasing intensity, reaches its maximum sooner than on low-pass filtration (Pollack 1948 b) There is no similar difficulty about noise in tests carried out with the low-pass filtration system, since because of the presence of high-energy vowels intensity need not be increased

The word material employed in this study consisted of familiar meaningful words In low-pass filtration distorted words are easier to guess (Miller and Nicely 1925) and this fact is apparently one of the reasons why the low end of the present low-pass curve lies on a higher level than that described e g by French and Steinberg (1947) and by Hirsh *et al* (1954) on investigations with meaningless syllables This is another factor affecting the site of intersection of the curves

b) BAND PASS FILTRATION

The purpose of the tests carried out with band pass filtration was to assess the importance of each frequency area for the intelligibility of Finnish speech, and to suggest from which area and in which width suitable bands for the binaural test could best be sought

A few initial tests were carried out using an attenuation of 30 db/octave to see whether bands for the binaural test could perhaps be found by such slight distortion A study of the results in Table 6 and Fig 16 shows that it might be possible, by suitable selection, to find a band for this purpose from the low end In the high end, however, discrimination became far too good in the frequency area suitable for use in the binaural test

In tests carried out with even such slight attenuation the typical distribution of discrimination in the frequency area is visible For the understanding of speech the frequencies around 1500—2000 cps seem quite clearly to be the most important This agrees with the results obtained by e g Fowler (1942, 1947), and by Bocca and Pellegrini (1950) Another area of importance for the intelligibility of speech is noted at around 400—600 cps

When a greater filter attenuation (60 db/octave) and bands of different widths are used, a much better idea is obtained of the frequency factors affecting the intelligibility of speech It is found that, when the narrow band is used, discrimination remains very poor throughout the frequency area and exceeds 20 per cent only in accidental cases (Fig 17) This seems quite natural for in the low frequency area in particular only one formant of each sound falls in such a narrow area Since even this formant is situated in different areas with different sounds, discrimination can be achieved only by a suitable combination of sounds

The fact that any intelligible speech whatever is produced on the narrow band is naturally a result of filter attenuation, which permits the frequencies outside the cut-off frequency to affect the intelligibility of the band Since the intensity of the band presented in these tests was at a sensation level of about 50 db, filter attenuation gives a band width of two-thirds of an octave at a sensation level of 30 db, where the frequencies concerned still make their maximum contribution to intelligibility (Miller 1951) According to Miller, a sensation level of even 15 db increases intelligibility by half the maximum, so that the effective width of the narrow band, using a 50 db sensation level, is about an octave This explains the high individual discrimination scores even when a narrow band was used Tests carried out with the other bands must also be judged against this background, so that the true band width is obtained by adding an octave to the band indicated by the cut off frequencies When this is taken into account, it seems a priori very probable that widening the bands by, say, 1/4 or 1/3 octave makes no decisive improvement in discrimination Even so, especially at the low end of the speech area, a rising tendency is seen in discrimination scores from 1/4 octave bands to 1/3 octave bands

When the bands are widened, a curve plotted through the points indicating mean discrimination is found to remain identical in shape up to the one-octave band (see Figs 16—20 and Fig 23) The curve has two marked peaks, a lower one at about 500—700 cps and a higher at about 1300—1700 cps Between these peaks, with a mean frequency of 700—900 cps, there is a pronounced depression The significance of this finding is increased by the fact that in binaurally effected tests, when bands taken from this depression are combined with the higher bands, the results obtained are systematically poorer than when bands of a mean frequency of 500—600 cps are combined with the higher bands Such a depression in the curve under discussion has not been described earlier In the study by Maspétiol and Semette (1956) there emerged, in a review of the individual values, a trend towards such discrimination scores, but limited material evidently caused the curve to be so extrapolated that the depression was not obvious

An explanation for this shape of curve comes from a study of the frequency structure of speech The lower peak coincides clearly with the first vowel formant, whereas there is a phonetically poor area at about 700—900 cps (see Fig 2 and Table 1) before, with increasing frequency, the second formants enter the band The maximum of the curve coincides with an area where, in addition to the second vowel formant, the frequencies exceeding 1300 cps, so important for the consonants, are also included

When the band is widened to cover one octave, the depression in the curve is smoothed out because the extension is so great that even if the mean frequency coincides with the depression area, the edges of the band absorb frequencies from both the first and the second formant Only after the band is widened to cover 1 ½ octaves can discrimination be made to rise in some cases to about 100 per cent between 1000 cps and 2000 cps Distortion, however, still produces so much individual difference that the mean value of discrimination in this area also falls below 80 per cent, although the first two formants of most vowels and numerous frequencies important for consonant discrimination are included

When the results obtained by means of low-pass and high pass filtration are surveyed and compared with those yielded by band-pass filtration, some observations can be made, particularly in areas where the low-pass and high-pass filtration curves fall close to zero It may be seen, at both the high and the low end, that while the cut-off value of high-pass or low-pass filtration yields a very poor discrimination score (about 10—20 per cent), a corresponding discrimination score is possible from a fairly narrow band in the frequency area concerned For example, low pass filtration with cut-off value 570 cps discrimination 8 7 ± 5 9 per cent, and 1 4 octave band 480—600 cps discrimination 13 7 ± 7 5 per cent This justifies the conclusion that the frequencies important for discrimination are contained within a very small area at both the high and the low end of the frequency area At the low end however there is a very important, and maybe decisive factor affecting this phenomenon which does not exist at the high end the band has an

advantage over low-pass filtered speech in that it lacks the high intensity low sounds which in this narrow area mask the frequencies that aid discrimination.

When the above described tests, carried out on individual bands are reviewed from the point of view of developing a binaural filtered speech test, it may be said that suitable bands can be sought from the high end at around 1200—3000 cps, primarily from among the narrow, 1/4 and 1/3 octave wide bands. In the low frequency area (400—1000 cps) a 1/2 octave wide band might also be considered, since its discrimination scores remain fairly low, especially if the word material is suitably selected.

## 2. BINAURAL TESTS

The intention was to ascertain, with binaural tests using phonetically balanced word material, the discrimination scores of various band combinations which might possibly be considered for a filtered speech test similar to Matzker's binaural test. The prime object of these tests was to discover whether some band combinations were definitely more suitable than others or whether greater freedom was possible in the selection of bands.

A large number of different band combinations had to be studied. The results of the monaural tests described above could be utilized in selecting the bands to be tested, at least in so far as bands providing too good discrimination could be excluded. Similarly the bands affected markedly by peripheral cochlear processes, such as acoustic trauma, could be excluded from the study. Bands extending much beyond 3000 cps were thus not worth examination. The bands sought had to meet at least the following requirements: discrimination when one band was used should not exceed 40 per cent, the combined discrimination of any pair of bands had to be at least in the region of 70 per cent, the bands should not contain many identical phonetical elements, and should therefore lie far enough apart bearing in mind the attenuation of the filter.

The search began among the narrowest possible bands where the discrimination score of one band would in no case be too high. Tests performed on a few test subjects revealed, however, that although a pair of bands providing the best possible discrimination was used (570 cps and 1800 cps), discrimination could only exceptionally be raised above 50 per cent. Widening the band to 1/4 octave immediately gave discrimination scores that looked more useful. A study of the results given in Figs 24—28 and a comparison with those given in Fig. 18 on the discrimination scores of 1/4 octave bands alone show that the bulk of discrimination with this band pair is based on the high band. Only for the highest band employed, 2400—2800 cps, was discrimination poorer than the best low bands. The low bands nevertheless decidedly improved discrimination.

# VIII FILTERED SPEECH TEST

The ultimate object of the present investigation was to develop a Finnish filtered speech test for routine detection of central hearing disorders The test should be so devised as to yield information varied enough using the simplest possible equipment over a relatively short period without strain on the patient The examiner's subjective estimation ought not to affect the test too much The patient's habituation to listening to and understanding filtered speech during the test ought to be eliminated Combination of the monaural and the binaural test would be useful It was felt that, if the following principles were adopted, a test meeting the above requirements might be devised

The basic test was to be a binaural synthesis of speech from two separate bands, on the principle of Matzker's test, and a monaural filtered speech test in which distortion was similar to that of the frequency bands employed in the binaural test On this point the present construction of the test corresponds to that described by Lindén (1960) In this way the results of binaural and monaural filtered speech tests should be more readily comparable than *e g* in Matzker's test, for the influence of a unilateral disorder on the binaural test is easier to assess If a word is discarded as erroneous when only one sound is incorrectly reproduced, the influence of the examiner's subjective evaluation is considerably reduced as compared with Matzker's test

To make the test short and simple it was important that the necessary information should be obtained by using only one intensity level This intensity should be strong enough to coincide not with the steeply rising part of the articulation curve but with the site where discrimination bordering on maximum is obtained This occurs when the intensity is raised to the sensation level of some 50 db A study of the results which Lindén, for example, obtained on brain tumour patients, shows that the necessary information is available even at the 50 db sensation level alone The same conclusion can be drawn from the observations published by Bocca *et al* (1955), Antonelli *et al* (1963 b), and by Calearo and Antonelli (1963). It also agrees with the general principles concerning the determination of discrimination scores in speech audiometry Raising the testing intensity to such a high level, however, necessitates special arrangements The interaural insulation has to be as good as possible, the best can be achieved by the use of insert earphones In band selection the discrimination provided by one band

did not have to be too high even at this high intensity level, careful selection of word material played its part here

The idea advanced by R Harris (1963) that in the binaural test every second word should be presented monaurally for the sake of control seemed very practical, especially if the monaural part was also presented alternately to each ear The outcome would be a test in which consecutive words were heard filtered first into the right, then into the left ear, and finally binaurally In this way previous acquaintance with filtered speech is not required, since all three parts of the test are fully comparable This also helps to shorten the test. At the same time it is possible to avoid a primarily psychological source of error pointed out by Harris if utterly poor discrimination is obtained under any one of the test conditions and this alone is used, the patient may be put off by the feeling that the test is altogether too difficult But if at intervals he hears words easier to understand and if, moreover, he must devote his attention alternately to each ear and to both ears, his interest is maintained, which is an important factor in the success of the test For the same reason it is advisable to include among the test words a few that are easier than the rest

## 1 SELECTION OF VOCABULARY AND BANDS

In the selection of vocabulary and bands the object was to find conditions which met the following requirements

1 The vocabulary should be one in which the mistakes most frequently encountered in normal material were eliminated, and which would provide the most favourable results possible in the filtering conditions chosen.

2 The binaural discrimination score of the two bands should be slightly below 80 per cent, in order that the same combination of bands would also serve for a monaural filtered speech test

3 The discrimination score for each single band should be below 30 per cent

4 The bands should not contain much common phonetic material and thus should be sufficiently wide apart, taking into account the attenuation of the filters and the intensity level employed.

The experiments already described justify the conclusion that, at least if phonetically balanced test material is employed, it is not possible to indicate a single pair of bands more favourable than the others to suit the test, but that they are best selected from the low end, 1/2 octave wide, between 480—840 cps, and from the high end, 1/3 octave wide, between 1560—2880 cps. In order that the phonetic material of the bands be sufficiently different, bands for continued study were selected as far apart as possible The highest band, however, which extended to 2880 cps, was not used since it came too close to the region in which hearing, for peripheral

reasons, may be locally reduced The bands finally selected were thus the low one of 480—720 cps and the high one of 1800—2400 cps

An analysis of the tests described above, carried out both monaurally and binaurally in these frequency areas, revealed that they contained a large number of words which were incorrectly reproduced in almost all distortion situations It was obvious that these words produced an unnecessary reduction in discrimination, and they had to be discarded For this reason the words selected were those which were understood in a minimum of 75 per cent of the tests in the binaural study of the above two bands These words were *kengan, tusina, jyra, meni, potilas, keha, puree, kaunis, virma, loydan, vilkkuu, lypsaa, oikku, peukalo, jyva, maistuu, häijy, silloin, julma, yossa, etta, nuoska, riepu, paukkuu, miten, maksu, pare, liukas, suunnaton, kellui, kaytos, meri, varis, kaden, syoda, kevat, sulaa, rumu, savel kuppi, sangon, ruoste, lapi, hiekka, vuori, pettaa, alku, soittaa, luja, kaivaa, saali, myyda, lappi, komea, nuori, kuljen, tuuli, seppa, muoti, sydan, soutaa, pelko, rauta, iskee, ruuti, koyha, myohaan, satoi, kiuru, oja, kylla*

The next step was to pick out the words which, from the high band alone, gave a correct result in over 50 per cent of the tests They were *syoda, kevat, rumu, ruoste, hiekka, sydan, savel, riepu, ruuti, kengan, meri, saali, virma*

The same discrimination score from the low band alone was produced by *varis, vuori, nuori, sydan, rauta, satoi, paukkuu, valo, alku, meri*

These two sets of words, understood from one band, were discarded when the final test vocabulary was planned There remained therefore only 50 words in the list of words understood in 75 per cent of the tests On the basis of these words another 70 phonetically similar words were chosen, by which means a list of 120 words was again obtained This list was tested separately using the low (480—720 cps) and the high (1800—2400 cps) bands and using both bands together Twenty subjects were employed for each of the three testing conditions, and listened to the whole 120 word list under one of the described distortion conditions It was found that the discrimination scores on the whole remained sufficiently low although some persons obtained remarkably high scores even from one band The 480—720 cps band alone gave an average discrimination score of $19\,1 \pm 9\,7$ per cent, while individual scores ranged from 7 to 42 per cent The discrimination score from the 1800—2400 cps band was $17\,9 \pm 11\,8$ per cent, range of variation being from 3 to 46 per cent The binaural discrimination score using these bands was $72\,4 \pm 14\,2$ per cent, the range being from 50 to 92 per cent

In the course of these tests the errors in individual words were noted in each test condition Using this information, 90 words that gave the best result could be selected from the 120 word list for the final test These words were divided into three lists in such a way that the intelligibility of each list on the basis of the above tests, was made as nearly identical as possible when tested on both bands separately and on the two together At the same time an attempt was made to make the lists phonetically

TABLE 17

*Word lists for the filtered speech test*

| List I | List II | List III |
|---|---|---|
| peukalo | rusina | sotilas |
| kiuru | loydan | vilkkuu |
| kellui | suljen | luoti |
| näytös | sitoi | paistuu |
| myyda | kaivo | leppa |
| tietää | puree | julma |
| että | muita | tuuli |
| jyra | naula | suoja |
| milloin | tyossä | selko |
| valjyys | juosta | luja |
| lappi | myohään | viulu |
| tusina | pelko | sulaa |
| koyha | tuuri | kuppi |
| nuoska | silloin | meilla |
| seppa | pyydan | paksu |
| maksu | heitto | pare |
| kaulus | potilas | siella |
| kiuas | peli | jyva |
| nöyra | tangon | kuja |
| tuoli | koivu | luoja |
| soittaa | nauris | noutaa |
| kohina | kiulu | kylla |
| tyohon | kaatui | hoyla |
| sangon | kypsä | naytos |
| komea | soutaa | leuka |
| keha | pukee | vasyi |
| moni | nuppi | kangen |
| maistuu | suomi | poydan |
| kauha | pettaä | voittaa |
| kaunis | vaijyy | tukee |

identical Each list was compiled to open with a number of easy words, since at the outset the listener was least accustomed to listening to filtered speech and most easily made mistakes In this way the three lists of words given in Table 17 were arrived at

In order to be able to compare in detail the lists obtained they were dictated on tape recorder, starting with the first word of each list, at 5 second intervals, followed by the second word, and so on, which gave a list of words that began as follows *peukalo, rusina, sotilas, kiuru, loydan, vilkkuu* This arrangement made each list equally difficult to learn during the test

The discrimination scores of the lists so dictated were subsequently tested once more, separately from both bands and together binaurally, using 60 test subjects, the results obtained are given in Table 18 It was found that even though the differences in mean values between the lists of words were not

## TABLE 18

*Discrimination scores for word lists I II and III in the filtered speech test, measured with three distortions each on 20 subjects*

| Band cps | Mean ± S.D List I | List II | List III | Total |
|---|---|---|---|---|
| 480—720 monaural | 16.0 ± 10.3 | 18.9 ± 10.2 | 17.4 ± 7.4 | 17.5 ± 7.9 |
| 1800—2400 monaural | 16.2 ± 7.3 | 16.8 ± 12.0 | 14.1 ± 9.9 | 15.6 ± 9.5 |
| 480—720 / 1800—2400 binaural | 66.6 ± 10.5 | 70.5 ± 10.7 | 69.2 ± 13.1 | 68.7 ± 11.0 |

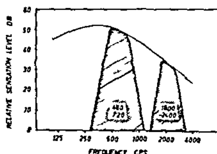



Fig 39

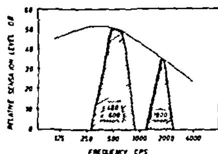



Fig 40

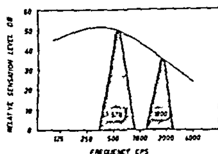



Fig 41

*Figs 39—41* Bands used in filtered speech test The curve at the very top of the picture shows the shape of the speech spectrum, idealized after French and Steinberg (1947)

statistically significant, an individual could show a 20 per cent difference between discrimination scores for the different lists

In order to eliminate the effect of the differences between the word lists in the test, the test was extended so that each object — the right ear, left ear and binaural hearing — was tested with each of the three lists of words Thus the number of test words in each test condition grew to 90, adding to the reliability of the results and making them independent of the differences between test words Admittedly, the time taken to perform the test was thereby trebled to about 25 minutes.

In these tests the wide individual variation emerged again A test with the bands concerned (480—720 cps and 1800—2400 cps) was evidently too easy for some subjects, since their discrimination scores exceeded 90 per cent For an effective test the distortion should be so strong as to reduce the discrimination scores of normal subjects to 60—80 per cent (Bocca 1956) This was the case with the majority of those tested Since it would be useful to be able to repeat the test, possibly several times, fairly soon after the first occasion, and since learning would here result in improved discrimination scores, it was considered necessary to supplement the test material with versions more difficult to understand This was achieved by narrowing the bands

When the bands were narrowed, the discrimination scores from the narrow bands of 570 cps and 1800 cps were first tested with the word material selected for the filtered speech test, individually and in combination, using 20 test subjects for each test condition The 570 cps band gave a discrimination score of 7 8 ± 7 3 per cent, with a range of variation from 0 to 18 per cent, and the 1800 cps band one of 12 9 ± 8 3 per cent, with a range of variation from 0 to 28 per cent The discrimination provided by these bands together was 54 2 ± 14 0 per cent, range from 27 to 74 per cent Hence even this pair of bands might be considered for some subjects in order to ensure distortion strong enough

The second new pair of bands selected was one that gave a slightly better discrimination the low band of 480—600 cps and the narrow high band of 1800 cps, which, judged on the basis of the foregoing tests, should provide roughly similar discrimination scores The intelligibility scored for the low band was 13 5 ± 6 7 per cent, the range being from 0 to 26 per cent, and for the two bands together 60 8 ± 16 0, the range being from 35 to 90 per cent Twenty test subjects were now tested for each test condition

For a fairly large number of subjects the discrimination was thus reduced to a suitable level, using the last-mentioned combination of bands For people with some experience of listening to distorted speech it is particularly advisable to choose for the test one or other of these stronger distortions The test was therefore recorded on tape, by the method to be described below, with these three degrees of difficulty, to provide in advance for individual variations When the test is repeated it may also be useful to change to more difficult test material

The bands employed in the filtered speech test are shown in Figs 39—41 Band intensities are here given in relation to the spectrum of speech in different frequency areas as they were prior to the equalization of sensation levels by which the intensity of even the high band was raised to the sensation level of 50 db The effective parts of the bands remain even then as illustrated (Figs 39—41), since when the high band is amplified the background noise of the equipment also becomes proportionately louder and as a result, the ratio of test sound to noise remains constant

## 2 TAPE-RECORDING OF TEST MATERIAL

The intention was to have the material tape-recorded, so that its presentation in the following order in routine testing should be as easy as possible the words of List I, using both the low and the high band, to one ear, and those of List II, also using the two bands, to the other ear, and the words of List III binaurally, using the low band for one and the high band for the other ear After each word test conditions would be changed in the following order monaural test to the right ear, monaural test to the left ear, binaural test In order that each of the lists would be presented by each of these three tests the lists were to be tape-recorded twice again, exchanging the order of presentation in a suitable manner

This test arrangement was simply achieved with the aid of a two-channel tape-recorder intended for stereo recording The three lists of words obtained as described above had been dictated on to tape so that the words came in turn from each list at five second intervals The vocabulary was fed through the equipment shown in Fig 7, in this way suitable bands could be fed, at the desired intensity, into the two channel tape recorder which was substituted for earphones By means of an audiometer serving as an attenuator the signals could be fed into tape-recorder channels in the order desired, either separately or in combination The first word was fed using both bands, to channel 1, the second word, also using both bands, was fed to channel 2, and the third word in such a way that the low band went to channel 1 and the high band to channel 2, the furth word, using both bands went to channel 1, and so on, until all three 30 word lists had been recorded The recording was then repeated in such a way that the words of List I went into channel 2 using both bands, those of List II with the low band to channel 1 and the high band to channel 2, and the words of List III to channel 1 with both bands The third recording was so effected that List I words went with the low band to channel 1 and with the high band to channel 2, List II words with both bands to channel 1 and List III words similarly with both bands to channel 2 Recordings were made using all three distortions described earlier (480—720 cps and 1800—2400 cps 480—600 cps and 1800 cps narrow, and 570 cps narrow and 1800 cps narrow)

The purpose of this recording method was to create circumstances in which when one ear listened to channel 1, and the other to channel 2 the testing conditions were varied, as required, automatically after each word and in such a way that each of the word lists was tested with each of the three test objects, the right ear, the left ear, and binaurally By feeding the vocabulary into the audiometer, by which the intensity of the channels could be adjusted as required, the equipment needed for a routine examination was ready for use The final equipment thus consisted of the tape containing the test material, a two-channel tape-recorder, and a two-channel audiometer with insert earphones

TABLE 19

*Discrimination scores obtained in the filtered speech test, measured with three distortions each on 20 subjects*

| Bands cps | Right ear | | Left ear | | Binaural | |
|---|---|---|---|---|---|---|
| | Mean ± S.D | Range | Mean ± S.D | Range | Mean ± S D | Range |
| 480—720<br>1800—2400 | 78.3 ± 7 4 | 64—88 | 80.1 ± 8.3 | 68—91 | 79.8 ± 7 1 | 69—90 |
| 480—600<br>1800 narrow | 65.2 ± 6.6 | 54—76 | 68.0 ± 9 7 | 50—80 | 66 8 ± 7 7 | 57—76 |
| 570 narrow<br>1800 narrow | 56.2 ± 12.1 | 36—74 | 57 1 ± 10.9 | 33—80 | 57 1 ± 10.9 | 33—80 |

## 3 THE TEST AND ITS RESULTS WITH NORMAL SUBJECTS

With the equipment described above the final filtered speech test was in practice easy to carry out. The intensity of both channels was adjusted by audiometer to a sensation level of 50 db, and the tape containing the test vocabulary was set running. Test conditions were varied automatically in the way required by means of the recording method described The results were entered in diary designed for the test, each of the three test objects had its own column in which the discrimination of each word was entered just as it usually is in speech audiometric tests The result was three figures which showed the number of mistakes in each of the three test objects, first the right ear, then the left ear, and finally binaurally For each test object, discrimination was tested using 90 words The final result was the precentage of correctly repeated words

This test was carried out on 60 healthy subjects with normal hearing The results are given in Table 19 The same table also gives the results of the tests carried out with two more difficult distortions

The results show that the scores of different subjects in a test carried out with the same distortion for all of them may differ considerably, up to 40 per cent, whereas the scores of one and the same subject in all three test conditions (right ear, left ear, binaural test) did not differ by more than 15 per cent, and usually by only about 5 per cent

## 4 EFFECT OF REDUCED PURE TONE THRESHOLD ON TESTING TECHNIQUE

The risk of overhearing limits the use of a filtered speech test for patients whose hearing is reduced for peripheral reasons

If there is a conductive hearing defect, the testing intensity in each ear must not exceed the 70 db monaural sensation level measured by the normal

ear, in order to avoid possible errors produced by overhearing If the degree of conductive defect exceeds 20 db, the test cannot be safely carried out at a sensation level of 50 db If the patient has a bilateral conductive defect of e g 30 db, the intensity, to reach 50 db sensation level must be raised to 80 db as compared with the normal ear This will give a 50 db sensation level in the ear to be tested, and a 10 db sensation level in the contralateral ear as a result of overhearing Even this small degree of overhearing may affect discrimination, for according to Miller (1951) a band of even 6 db sensation level increases intelligibility by one-fifth of its maximal addition to discrimination score In the monaural test in particular this obviously increases discrimination, but in the binaural test it may happen that, particularly if the weaker, overheard band is high, the low band of considerably greater intensity given to the same ear masks it so that it does not affect discrimination The situation is in principle the same as that described by Bocca (see p 15), except that there are now two fractions of speech with frequency areas differing more considerably The effect of this is being studied

In order to avoid the drawbacks caused by overhearing the test can, if required, be carried out using a sensation level below 50 db, down to about 30 db, provided that the discrimination-reducing effect is taken into account when the results of the test are appraised Because of the construction of the test the lowering of sensation level has a similar effect on all three test objects if there has been no overhearing To obtain a normal control value for such a situation the test was carried out, using a sensation level of 30 db, on 20 healthy subjects of normal hearing The results obtained were right ear 64 8 ± 14 5 per cent, left ear 66 3 ± 15 1 per cent, binaural test 64 2 ± 14 8 per cent, individual values ranged from 41 to 84 per cent, 32 to 88 per cent, and 32 to 80 per cent, respectively

These results were obtained using minimum distortion, in other words bands 480—720 cps and 1800—2400 cps This is to be recommended whenever *the test is carried out at a sensation level below 50 db*

In perceptive hearing defects the above again holds good in that the sound intensity presented to the poorer ear in unilateral defects must not exceed the 70 db sensation level of the better ear In bilateral perceptive defects it is possible to employ sensation levels exceeding 70 db for the normal ear, because the perceptive defect reduces the danger of overhearing

For combined hearing defects the rules to be followed are generally the same as for conductive defects The intensity may, however, be raised in some cases by as many decibels as the bone conduction is reduced in the ear with better cochlear function

The effect of cochlear defects on the filtered speech test is being studied

## 5 CONCLUSION

The filtered speech test described above meets all the requirements which earlier studies in different parts of the world have shown to be necessary in the evaluation of central hearing disorders A distortion giving the necessary 60—80 per cent discrimination in a normal series is created by this test, so also are the conditions under which the results obtained for both ears are equivalent and comparable Hence the requirements for a monaural test are met Binaurally, the present test results in a low discrimination score for the two bands separately but in a high score for two combined Since, moreover, overhearing is eliminated, the basic conditions for a successful binaural test exist One of the advantages of the present test is that the binaural part of the test can be checked by means of the comparable results given separately for each ear Another advantage is that the test can be repeated immediately and frequently, if required, using the same word material, without any deterioration in the results If discrimination on repeated tests tends to rise too high the discrimination level can be adjusted by changing the distortion, without affecting the mutual relations of the test objects As the equipment is simple, the method seems well adapted for routine use

Studies of the results which this test gives in various central defects are now in progress

affecting the interpretation of the results The level of discrimination can be adjusted, if required, by changing the bands In tests carried out on 60 normal test subjects each of the three test objects gave nearly the same result, the range of variation between the test objects for one and the same test subject never exceeded 15 per cent The test results are given in Table 19

Studies of the results this test gives in various central and peripheral disorders are in progress

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S U P P L E M E N T U M 211

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## ABSTRACT

The threshold of hearing of each ear of 178 soldiers was measured before and after various types of shoulder rifles were fired at the rate of one trigger pull every five seconds An evaluation (peak pressure, time history, and spectrum) of the acoustic impulse from each type of weapon was performed The peak pressures of the acoustic impulses were highly correlated with temporary threshold shifts of hearing resulting from exposure to the gun noise Estimates are made, on the basis of the presently obtained and related data, of the expected permanent hearing level in the frequency region from 1000 to 6000 cps to be equaled or exceeded in 50%, 25%, and 10% of ears repeatedly exposed to gun noise at various peak sound pressure levels

## INTRODUCTION

There is ample evidence that the noise from firearms can cause various degrees of hearing loss in some persons exposed to the noise (4, 5, 6, 8, 9, 16–21) Although the articles report clinical observations and post-exposure audiometric measurements of the damaging effects of gun noise on hearing it is difficult to determine from these data the correlation between the physical characteristics of gun noise and the degree of hearing loss suffered by persons exposed to the noise

A recent program of tests designed to evaluate some existing and proposed small arms of the U.S Army provided an opportunity to obtain under relatively controlled conditions further data on the problem of auditory fatigue and damage due to gun noise Further, it seemed possible that if properly carried out, the tests could lead to conclusions concerning damage risk to hearing that might be helpful for the establishment of criteria for exposure to gun noise in general To that end, a series of tests involving the measurement of the physical characteristics of the noise and the effects of such noise on the threshold of audibility of the persons firing the weapons was undertaken

## PROCEDURE—AUDITORY TESTS

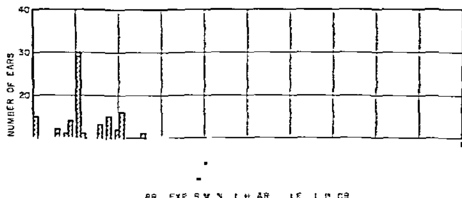
Prior to the start of the weapon-evaluation tests, some 178 infantrymen were given a series of four pure-tone audiograms under laboratory conditions using a Rudmose ARJ IV automatic audiometer These pre-experi-

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*Fi 1 Pre-experimental hearing level at 3000 cps of all ears tested Unshaded portion represents ears eliminated from the study*

procedures described in Ref 10 The maximum time difference between any measured TTS and two minutes post-exposure was four minutes

The hearing level (HL) values determined from the audiograms are in terms of the ASA 1951 Standard (2)

## Pre Experimental Hearing Levels

The average of the pre experimental hearing level for each test frequency (500 1000 2000 3000 4000 and 6000 cps) revealed that although a number of the subjects had normal or better than normal hearing, according to present ASA specifications (2) the distribution of HL s was markedly skewed Figure 1 shows by example for the 3000 cps test frequency that the thresholds of audibility of a sizable percentage of the subjects are considerably below the usual range of normal This finding is in agreement with previous hearing tests of U S Army personnel (6 12)

## Data Eliminated From Study

Although all the persons given pre-experimental audiograms participated in the firing of the weapons the audiometric data obtained later for some of the ears were excluded from our analysis of the effects of firing on hearing The ears eliminated from consideration were those having pre-exposure HL s greater than 30 dB at 2000 cps or 40 dB at 3000 cps This was done because (a) ears with this amount of pre-exposure hearing loss presumably permanent—could hardly be expected to show any shifts in threshold as the result of the noise exposures planned for this experiment it was planned to limit exposures to gun noise such that the average amount of TTS would be hopefully less than 30 dB at 2000 cps and 40 dB

TABLE 1 *Scheduled firing conditions*

| Weapon | Number of trigger pulls/number of rounds per trigger pull | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 15/1 | 30/1 | 60/1 | 100/1 | 50/2 | 8/2 | 100/2 | 10/3 | 20/3 | 30/3 |
| A | × | × | × | × | | | | × | × | × |
| B | × | × | × | × | × | × | × | | | |
| C | | × | | × | | | | | | |
| D | | | | × | | | | | | |

*Note* Due to some misfires on Weapon A and Weapon C the actual number of rounds fired and the interval between some rounds differed somewhat from that planned (see Table 2)

mental audiograms were administered to familiarize the subjects with the audiometric procedures and to provide an accurate measure of the threshold of hearing for each subject In addition audiograms were obtained at the firing range before and after all firings of the guns by the infantrymen

Five Rudmose ARJ-4 audiometers with otocups (special sound insulating cushion that covers the audiometer earphones) were used at the firing range Audiograms were administered with the subject seated in a portable metal booth approximately 30 feet from the firing line Octave band sound pressure level measurements revealed that the noise levels inside the booths were less than the tolerable levels for audiometric booths as specified by ASA (1) the wearing of otocups further ensured that the audiograms were given without any noise interference or masking

Immediately prior to any firing of the weapons, five subjects were given a pre-exposure hearing test in the test booths The subjects then moved up to the firing line and assumed a prone position the men were positioned approximately 20 feet apart Every five seconds on signal from a whistle the five men would pull the trigger of the weapon once After a designated number of trigger pulls the subjects immediately moved back to their respective audiometer The hearing test was begun approximately 15 seconds after firing the last round All subjects were tested in the same manner The firing conditions that were employed are shown in Table 1

The difference if any between the pre- and post-exposure audiograms at each test frequency called the temporary threshold shift (TTS) was determined after each firing sequence Because the auditory threshold normally returns to pre-exposure levels so rapidly it is customary to report

... unt of threshold shift present exactly two minutes after exposure

... was not possible to measure each ear at each audiometric

... tly at two minutes postexposure it was necessary to

... e thresholds measured to TTS in accordance with

# RESULTS OF PRE- AND POST-EXPOSURE AUDITORY TESTS

## Hearing Levels for Near Ear and Far Ear

It would seem reasonable that the ear nearest the muzzle of a rifle (the left ear of a person firing right handed, and vice-versa) would tend to have a higher hearing level than the other or "far ear", inasmuch as acoustical measurements indicate that the sound-pressure level from a weapon is usually about 1 dB greater at the "near ear" than at the "far ear" Pre-exposure HL's and $TTS_2$'s for the near ear and far ear are shown in Table 3 for the various sub groups of subjects It is seen that the expected difference between near-ear and far-ear hearing does not seem to be consistently present in these results (see also Collins (5))

The near ear may indeed suffer more abuse from gun noise than the far ear, but these particular ears did not show this difference, possibly because they had suffered exposure to other noise sources in the past that were more damaging to their far ear (for small weapon firing), than to their near ear In any event, we considered each ear as a separate "subject", and in the remaining presentations of data the results obtained on each ear of an individual are included as separate datum points

## Summary of Data

Table 4 presents a summary of statistical analyses of the audiometric data obtained before and after exposure of the subjects to the various firing conditions, the average, the 25th ($Q_1$), 50th (median or $Q_2$), and 75th ($Q_3$) percentiles, and the number of ears are given for most of the firing conditions $Q_1$, $Q_2$, and $Q_3$ values were not determined for some of the firing conditions which caused but small threshold shifts

Arguments can be made for the use of either the average or the median as a description of the central tendency of the data at hand The average seems the more logical if we wish to answer the question as to what is the *amount* of threshold shift, experienced by the group as the result of exposure to a given firing condition, on the other hand, the median should tell us the level of hearing loss or threshold shift that would be exceeded by 50 per cent of the exposed population However, the number of ears involved in some of the sub-groups of subjects made these statistics, occasionally, rather unreliable

## $TTS_2$ Results

We examined both the average and median $TTS_2$ values for each of the various firings conditions as a function of audiometer test frequency

TABLE 2 *Average and median pre exposure hearing levels, averaged at 1000, 2000, and 3000 cps*

| Weapon | No. of trigger pulls/No. of rounds per pull | No. of ears | Average | Median |
|---|---|---|---|---|
| A | 17/1 | 9 | 0.7 | 1.3 |
| | 32/1 | 18 | 6.0 | 5.0 |
| | 74/1 | 6 | 3.2 | 1.0 |
| | 102/1 | 6 | 2.1 | 4.0 |
| | 6/3 | 6 | 2.4 | 4.3 |
| | 13/3 | 3 | 5.8 | 5.0 |
| | 21/3 | 19 | 2.0 | −0.7 |
| | 31/3 | 7 | 1.2 | 0.3 |
| B | 15/1 | 16 | 6.4 | 6.7 |
| | 30/1 | 21 | 3.6 | 2.0 |
| | 60/1 | 35 | 2.3 | 1.3 |
| | 100/1 | 36 | 2.7 | 2.0 |
| | 50/2 | 17 | −0.9 | −1.3 |
| | 85/2 | 11 | 0 | −2.0 |
| | 100/2 | 23 | 2.5 | 1.7 |
| C | 23/1 | 5 | 4.1 | 3.7 |
| | 63/1 | 12 | 3.8 | 1.0 |
| | 97/1 | 27 | 2.9 | 3.3 |
| D | 100/1 | 30 | 1.0 | −1.3 |

at 3000 cps), and (b) the relatively few ears with these large HL's were not equally distributed among the various firing conditions and their HL's introduced a relatively strong and unequal bias in the audiometer test results, particularly in the average results for the various firing conditions. A total of 55 ears were thus eliminated from our test population of 356 ears.

Although the hearing level at 500 cps was determined for each ear before and after each exposure to the gun noise, these data were excluded because (a) precursory examination of the audiometric results indicated that there were no consistent threshold shifts at 500 cps as the result of exposure to any of the firing conditions, and (b) the audiograms at 500 cps, which were always taken first in each audiometric test session, tended to be much more variable than those for the higher test frequencies.

## PRE-EXPOSURE HEARING LEVELS

The average and median pre-exposure HL's were remarkably similar for the different groups of subjects used for the various firing conditions. That these groups were, on the average, about equal is clearly shown in Table 2, where the average of the average and median values found at 1000, 2000, and 3000 cps are presented.

# RESULTS OF PRE- AND POST-EXPOSURE AUDITORY TESTS

## Hearing Levels for Near Ear and Far Ear

It would seem reasonable that the ear nearest the muzzle of a rifle (the left ear of a person firing right handed, and vice-versa) would tend to have a higher hearing level than the other or "far ear", inasmuch as acoustical measurements indicate that the sound-pressure level from a weapon is usually about 1 dB greater at the "near ear" than at the "far ear". Pre-exposure HL's and $TTS_2$'s for the near ear and far ear are shown in Table 3 for the various sub-groups of subjects It is seen that the expected difference between near-ear and far-ear hearing does not seem to be consistently present in these results (see also Collins (5))

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## $TTS_2$ Results

We examined both the average and median $TTS_2$ values for each of the various firings conditions as a function of audiometer test frequency

TABLE 3 *Comparison of Near Ear (NE) and Far Ear (FE) $TTS_2$ and pre exposure hearing level (mean) for those firing conditions causing the larger TTS's with each type of weapon*

| | $TTS_2$ | | Pre-exposure HL | |
|---|---|---|---|---|
| | NE | FE | NE | FE |
| **Weapon A 32/1 (N = 9)** | | | | |
| 1000 | 4.6 | 4.0 | 2.7 | 3.7 |
| 2000 | 7.7 | 9.1 | 1.2 | 6.2 |
| 3000 | 8.9 | 19.5 | 8.6 | 13.8 |
| 4000 | 15.1 | 16.1 | 11.2 | 25.1 |
| 6000 | 7.9 | 19.0 | 20.4 | 23.6 |
| Av | 8.8 | 13.5 | 8.9 | 11.5 |
| **Weapon A 74/1 (N = 3)** | | | | |
| 1000 | 10.0 | 7.5 | 5.3 | 2.0 |
| 2000 | 24.3 | 27.3 | 2.3 | −1.3 |
| 3000 | 23.7 | 57.5 | 12.0 | −1.5 |
| 4000 | 20.7 | 44.0 | 18.0 | 8.5 |
| 6000 | 40.0 | 51.3 | 11.7 | −4.5 |
| Av | 23.0 | 42.0 | 10.5 | 0.4 |
| **Weapon A 102/1 (N = 3)** | | | | |
| 1000 | 11.3 | 15.8 | 1.7 | 0.3 |
| 2000 | 28.7 | 33.5 | 0.3 | 1.0 |
| 3000 | [illegible] | 32.1 | 1.3 | 10.8 |
| 4000 | 17.3 | 33.1 | 1.0 | 21.8 |
| 6000 | 29.3 | 26.8 | 22.7 | 21.4 |
| Av | [illegible] | 28.1 | 4.8 | 11.5 |
| **Weapon B 100/1 (N = 18)** | | | | |
| 1000 | 3 | 4 | 2 | 2 |
| 2000 | 5 | 13 | 2 | 1 |
| 3000 | 19 | 15 | 8 | 5 |
| 4000 | 18 | 19 | 17 | 8 |
| 6000 | 28 | 16 | 17 | 13 |
| Av | 11.6 | 13.4 | 9.2 | 6.6 |
| **Weapon B 85/2 (N = 5)** | | | | |
| 1000 | 0 | 1 | 3 | 4 |
| 2000 | 0 | 5 | 4 | 5 |
| 3000 | 6 | 14 | 11 | 6 |
| 4000 | 14 | 21 | 8 | 16 |
| 6000 | 14 | 5 | 10 | 13 |
| Av | 7.8 | 9.8 | 6.0 | 3.6 |
| **Weapon B 100/2 (N = 11)** | | | | |
| 1000 | 2 | 3 | 3 | 0 |
| 2000 | 8 | 7 | 2 | −1 |
| 3000 | 13 | 12 | 5 | 6 |
| 4000 | 13 | 4 | 9 | 15 |
| 6000 | 10 | 3 | 15 | 16 |
| Av | 9.2 | 5.8 | 6.8 | 7.2 |
| **Weapon C 23/1 (N = 3)** | | | | |
| 1000 | −7 | 4 | 6 | 3 |
| 2000 | 2 | 9 | 1 | 4 |
| 3000 | 0 | 22 | 5 | 6 |
| 4000 | 2 | 31 | −2 | 4 |
| 6000 | 10 | 11 | 4 | 12 |
| Av | 1.4 | 22.0 | 2.8 | 5.6 |
| **Weapon C 63/1 (N = 6)** | | | | |
| 1000 | 4 | 6 | 4 | 5 |
| 2000 | 4 | 2 | 2 | 0 |
| 3000 | 11 | 9 | 8 | 2 |
| 4000 | 18 | 5 | 0 | 2 |
| 6000 | 23 | 5 | 12 | 19 |
| Av | 12.6 | 5.4 | 7.0 | 5.6 |
| **Weapon C 97/1 (N = 13)** | | | | |
| 1000 | 6 | 1 | 0 | 2 |
| 2000 | 10 | 3 | 1 | 3 |
| 3000 | 10 | 2 | 7 | 6 |
| 4000 | 12 | 5 | 11 | 7 |
| 6000 | 15 | 6 | 16 | 13 |
| Av | 10.4 | 3.0 | 6.6 | 6.2 |
| **Weapon D 106/1 (N = 15)** | | | | |
| 1000 | 2 | 2 | 0 | 2 |
| 2000 | 4 | 6 | 1 | 4 |
| 3000 | 8 | 5 | 4 | 0 |
| 4000 | 5 | 10 | 8 | 11 |
| 6000 | 8 | 8 | 17 | 14 |
| Av | 5.4 | 6.2 | 5.6 | 5.4 |

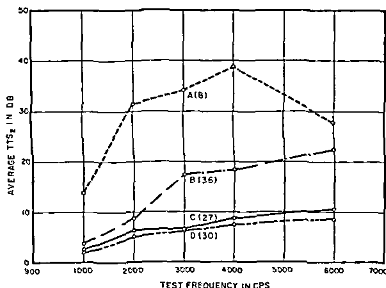



Fig 2 Average $TTS_2$ as a function of the audiometric test frequency following 97–102 trigger pulls of each weapon The number in parentheses indicates the number of ears averaged for each weapon

The averages seemed to give somewhat smoother functions than the medians, average $TTS_2$ values found when 97–102 trigger pulls were used are shown in Fig 2

## $HL_2$ RESULTS

In spite of the similarity of the ears for each group with respect to pre-exposure average and median HL's (Table 2), there were still present relatively large differences among the individual ears, as indicated by the size of $Q_3$ (Table 4) Therefore, an artifact would possibly be present in a direct comparison of the $TTS_2$ values found for the different firing conditions

Differences among individual ears with respect to their pre-exposure HL's would undoubtedly influence the amount of $TTS_2$ that was measured, for example, an exposure condition capable of causing a 15 dB TTS, in an ear, with a pre-exposure HL of 0 dB, should cause a much smaller, if any, $TTS_2$ in an ear with a pre-exposure HL of say 15 dB We have attempted to account to some extent for these individual differences in pre exposure HL's by adding together $TTS_2$ and pre-exposure HL to achieve "HL " or what the actual hearing level would be for an ear measured two minutes after exposure to a given noise condition

This procedure is to some extent supported by experimental evidence that the amount of $TTS_2$ suffered as the result of exposure to continuous steady-state noise is inversely proportional to their pre-exposure hearing

TABLE 4. Summary of data for pre-exposure hearing level, $TTS_2$ and $HL_2$

| | Pre-exposure hearing level 1 | 2 | 3 | 4 | 6 | $TTS_2$, Test frequencies in kc. 1 | 2 | 3 | 4 | 6 | $HL_2$ 1 | 2 | 3 | 4 | 6 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| [illegible] | | | | | | | | | | | | | | | |
| [illegible] (N=[illegible]) Average | 1.1 | 0.5 | 4.0 | 2.1 | 6.8 | 5.6 | 7.1 | 12.1 | 21.9 | 23.8 | 1.6 | 7.0 | 16.1 | 21.6 | 30.5 |
| $Q_1$ | 6.0 | 6.9 | 5.0 | 3.0 | 2.0 | 5.0 | 1.0 | 2.0 | 0 | 0 | 1.0 | 0 | 8.0 | 10.0 | 6.0 |
| Median $Q_2$ | 2.0 | 0 | 1.0 | 5.0 | 11.0 | 2.0 | 1.0 | 1.0 | 27.0 | 16.0 | 5.0 | 1.0 | 11.0 | 18.0 | 21.0 |
| $Q_3$ | 2.0 | 7.5 | 13.0 | 10.0 | 15.0 | 8.0 | 16.0 | 12.0 | 18.0 | 17.0 | 9.0 | 10.0 | 27.0 | 11.0 | 37.0 |
| 121 (N=18) Average | 3.2 | 7.7 | 11.2 | 18.2 | 22.6 | 4.3 | 9.1 | 13.9 | 15.6 | 11.1 | 7.5 | 12.1 | 25.1 | 31.8 | 35.1 |
| $Q_1$ | 3.0 | 1.5 | 1.0 | 2.0 | 9.0 | 0 | 2.0 | 2.0 | 2.0 | 0 | 2.0 | 5.0 | 5.0 | 11.0 | 12.0 |
| Median $Q_2$ | 1.0 | 1.5 | 11.0 | 21.0 | 21.0 | 5.0 | 5.0 | 5.0 | 6.0 | 7.0 | 5.0 | 6.0 | 20.0 | 27.0 | 40.0 |
| $Q_3$ | 10.0 | 15.0 | 14.0 | 31.0 | 31.0 | 6.0 | 12.0 | 21.0 | 25.0 | 40.0 | 10.0 | 16.0 | 27.0 | 60.0 | 57.0 |
| [illegible]41 (N=11) Average | 1.0 | 1.6 | 6.6 | 14.2 | 4.3 | 9.0 | 21.3 | 37.3 | 38.8 | 15.7 | 13.0 | 22.9 | 13.8 | 51.0 | 50.0 |
| $Q_1$ | 5.0 | 6.0 | 1.0 | 6.0 | 1.0 | 1.0 | 1.0 | 5.0 | 2.0 | 0 | 2.0 | 1.0 | 9.0 | 11.0 | 8.0 |
| Median $Q_2$ | 0 | 3.6 | 6.0 | 8.0 | 4.0 | 12.0 | 30.0 | 16.0 | 42.0 | 17.0 | 12.0 | 25.0 | 11.0 | 71.0 | 72.0 |
| $Q_3$ | 15.0 | 7.6 | 23.0 | 20.0 | 17.0 | 11.0 | 62.0 | 50.0 | 63.0 | 71.0 | 22.0 | 30.0 | 5.0 | 85.0 | 80.0 |
| 1721 (N=[illegible]) Average | 0.6 | 0.1 | 6.3 | 11.0 | 21.8 | 13.9 | 31.1 | 31.0 | 38.7 | 27.8 | 11.5 | 11.8 | 10.1 | 52.7 | 51.6 |
| $Q_1$ | 2.0 | 3.6 | 7.0 | 3.0 | 0 | 3.0 | 2.0 | 1.0 | 19.0 | 1.0 | 0 | 7.0 | 6.0 | 11.0 | 5.0 |
| Median $Q_2$ | 0 | 2.6 | 10.0 | 12.0 | 7.0 | 13.0 | 15.0 | 6.0 | 31.0 | 1.0 | 16.0 | 17.0 | 50.0 | 11.0 | 60.0 |
| $Q_3$ | 3.0 | 1.6 | 15.0 | 28.0 | 32.0 | 27.0 | 51.0 | 80.0 | 53.0 | 18.0 | 20.0 | 30.0 | 81.0 | 81.0 | 91.0 |
| [illegible]1 (N=11) Average | 3.0 | 0.3 | 1.1 | 11.2 | 7.0 | 2.8 | 3.8 | 8.8 | 7.3 | 1.8 | 5.8 | 1.5 | 13.2 | 18.5 | 11.8 |
| 131 (N=9) Average | 2.7 | 1.7 | 13.0 | 28.3 | 2.7 | 0.7 | 8.7 | 11.0 | 1.3 | 31.3 | 3.1 | 10.1 | 21.0 | 27.0 | 28.6 |
| 2[illegible]1 (N=10) Average | 0.1 | 2.8 | 1.1 | 6.1 | 13.6 | 3.6 | 1.1 | 6.1 | 6.7 | 1.8 | 2.7 | 7.2 | 10.5 | 11.1 | 18.2 |
| [illegible]1 (N=7) Average | 1.9 | 0.3 | 5.1 | 13.9 | 11.7 | 0 | 9.7 | 9.1 | 2.1 | 10.1 | −1.1 | 10.0 | 11.5 | 16.0 | 25.1 |
| [illegible] | | | | | | | | | | | | | | | |
| 151 (N=17) Average | 3.2 | 0.1 | 16.1 | 27.1 | 23.3 | 2.2 | 1.8 | 1.6 | 0.5 | 1.8 | 5.1 | 3.1 | 20.7 | 20.8 | 25.1 |
| 501 (N=21) Average | 1.7 | 2.3 | 6.7 | 17.1 | 21.1 | 0.2 | 2.3 | 1.5 | 1.6 | 1.5 | 1.5 | 1.6 | 11.2 | 17.7 | 27.9 |
| $Q_1$ | 1.0 | 3.6 | 1.0 | 0 | 7.0 | 3.0 | 1.0 | 1.0 | −1.0 | 2.0 | 2.0 | −1.0 | 1.0 | 1.0 | 6.0 |
| Median $Q_2$ | 0 | 1.6 | 5.0 | 9.0 | 19.0 | 1.0 | 1.0 | 1.0 | 3.0 | 3.0 | 1.0 | 1.0 | 6.0 | 10.0 | 21.0 |
| $Q_3$ | 5.0 | 8.6 | 17.0 | 21.0 | 13.0 | 3.0 | 1.0 | 8.0 | 12.0 | 10.0 | 6.0 | 9.0 | 21.0 | 21.0 | 55.0 |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 60/1 (*N* = 35) Average | 1.1 | −0.5 | 6.2 | 9.4 | 13.3 | 2.1 | 5.1 | 6.2 | 5.1 | 6.6 | 3.2 | 4.6 | 12.4 | 11.8 | 19.9 |
| $Q_1$ | −4.0 | −6.0 | −3.0 | 0 | 5.0 | −1.0 | 0 | 0 | −1.0 | −1.0 | −2.0 | −2.0 | 0 | −1.0 | 5.0 |
| Median $Q_2$ | 0 | −1.0 | 5.0 | 5.0 | 10.0 | 1.0 | 2.0 | 3.0 | 1.0 | 1.0 | 1.0 | 2.0 | 8.0 | 9.0 | 15.0 |
| $Q_3$ | 5.0 | 2.0 | 11.0 | 18.0 | 28.0 | 4.0 | 10.0 | 10.0 | 9.0 | 16.0 | 8.0 | 10.0 | 22.0 | 25.0 | 29.0 |
| 100/1 (*N* = 36) Average | 2.0 | 0.4 | 6.5 | 12.2 | 18.1 | 3.7 | 8.6 | 17.1 | 18.2 | 22.2 | 5.7 | 9.0 | 23.6 | 30.4 | 10.3 |
| $Q_1$ | −3.0 | −4.0 | −2.0 | −1.0 | −1.0 | 1.0 | 1.0 | 3.0 | 3.0 | 4.0 | 5.0 | 5.0 | 3.0 | 5.0 | 13.0 |
| Median $Q_2$ | 2.0 | 1.0 | 3.0 | 3.0 | 11.0 | 3.0 | 1.0 | 8.0 | 12.0 | 17.0 | −1.0 | 0 | 12.0 | 18.0 | 11.0 |
| $Q_3$ | 6.0 | 5.0 | 9.0 | 20.0 | 27.0 | 5.0 | 13.0 | 25.0 | 30.0 | 40.0 | 10.0 | 12.0 | 13.0 | 52.0 | 57.0 |
| 50/2 (*N* = 17) Average | 3.1 | −1.4 | 7.2 | 13.9 | 12.1 | 2.8 | 4.8 | 2.1 | 3.7 | 6.7 | −0.3 | 3.4 | 9.6 | 17.6 | 18.8 |
| 85/2 (*N* = 11) Average | −1.5 | 0.4 | 1.1 | 11.0 | 11.1 | 0.5 | 2.4 | 9.8 | 17.7 | 12.7 | −1.0 | 2.8 | 13.9 | 28.7 | 27.1 |
| 110/2 (*N* = 23) Average | 1.4 | 0.6 | 5.6 | 12.2 | 15.6 | 2.2 | 7.5 | 12.2 | 8.1 | 6.5 | 3.5 | 8.1 | 17.8 | 20.6 | 22.1 |
| *Weapon C* | | | | | | | | | | | | | | | |
| 24/1 (*N* = 5) Average | 4.0 | 2.6 | 5.6 | 1.2 | 9.0 | −0.2 | 6.0 | 13.0 | 21.4 | 28.2 | 3.8 | 8.6 | 18.6 | 22.6 | 37.2 |
| $Q_1$ | 2.0 | 0 | 0 | −3.0 | 0 | −8.0 | −1.0 | 3.0 | 2.0 | 8.0 | −3.0 | 0 | 9.0 | 0 | 10.0 |
| Median $Q_2$ | 4.0 | 0 | 7.0 | 0 | 1.0 | −3.0 | 1.0 | 11.0 | 4.0 | 13.0 | 3.0 | 2.0 | 19.0 | 40.0 | 59.0 |
| $Q_3$ | 7.0 | 7.0 | 11.0 | 7.0 | 21.0 | 0 | 3.0 | 11.0 | 50.0 | 56.0 | 12.0 | 18.0 | 51.0 | 65.0 | 73.0 |
| 63/1 (*N* = 12) Average | 2.9 | 1.6 | 8.9 | 13.4 | 13.1 | 1.8 | 3.3 | 11.7 | 13.0 | 15.4 | 7.7 | 4.9 | 18.6 | 20.4 | 28.5 |
| $Q_1$ | −1.0 | −5.0 | 0 | 0 | −3.0 | 0 | −1.0 | −2.0 | 1.0 | 1.0 | 5.0 | 0 | 5.0 | 1.0 | 6.0 |
| Median $Q_2$ | 2.0 | 3.0 | 7.0 | 9.0 | 9.0 | 3.0 | 5.0 | 1.0 | 11.0 | 8.0 | 7.0 | 5.0 | 10.0 | 32.0 | 19.0 |
| $Q_3$ | 8.0 | 6.0 | 11.0 | 29.0 | 15.0 | 9.0 | 8.0 | 10.0 | 30.0 | 35.0 | 11.0 | 12.0 | 38.0 | 55.0 | 56.0 |
| 97/1 (*N* = 27) Average | 1.1 | 0.7 | 6.5 | 9.1 | 11.7 | 2.1 | 6.2 | 6.2 | 8.7 | 10.1 | 3.8 | 6.9 | 12.7 | 17.8 | 25.0 |
| $Q_1$ | −5.0 | −5.0 | 0 | −3.0 | 1.0 | −2.0 | 2.0 | −1.0 | −2.0 | −2.0 | −2.0 | −2.0 | −3.0 | −1.0 | 7.0 |
| Median $Q_2$ | 1.0 | 5.0 | 4.0 | 6.0 | 12.0 | 1.0 | 1.0 | 2.0 | 7.0 | 1.0 | −2.0 | −1.0 | 8.0 | 1.0 | 2.0 |
| $Q_3$ | 5.0 | 13.0 | 13.0 | 11.0 | 20.0 | 9.0 | 9.0 | 11.0 | 20.0 | 22.0 | 7.0 | 10.0 | 12.0 | 14.0 | 27.0 |
| *Weapon D* | | | | | | | | | | | | | | | |
| 100/1 (*N* = 30) Average | −0.9 | −2.5 | 0.3 | 10.7 | 18.2 | 2.0 | 5.0 | 6.1 | 7.3 | 8.3 | 1.1 | 2.5 | 6.3 | 18.0 | 26.5 |
| $Q_1$ | −5.0 | −7.0 | −8.0 | −1.0 | 0 | −2.0 | 1.0 | 0 | 0 | 0 | 1.0 | 1.0 | −1.0 | 2.0 | 1.0 |
| Median $Q_2$ | −1.0 | −3.0 | 0 | 7.0 | 15.0 | 3.0 | 3.0 | 2.0 | 6.0 | 3.0 | 0 | 1.0 | 1.0 | 8.0 | 26.0 |
| $Q_3$ | 3.0 | 1.0 | 7.0 | 25.0 | 40.0 | 5.0 | 8.0 | 13.0 | 12.0 | 10.0 | 7.0 | 8.0 | 11.0 | 13.0 | 15.0 |

| Level | | | Pre-exposure hearing level | | | | | TTS$_2$ Test frequencies in KC | | | | | [illegible] | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 1 | 2 | 3 | 4 | 6 | 1 | 2 | 3 | 4 | 6 | 1 | 2 | 3 | 4 | 6 |
| *Weapon A* | | | | | | | | | | | | | | | | | |
| 17/1 | ($N$ = 9) | Average | —1 4 | —0 5 | 1 0 | 2 9 | 6 8 | 5 6 | 7 4 | 12 1 | 21 9 | 23 8 | 3 6 | 7 0 | 16 1 | 24 6 | 30 5 |
| | | $Q_1$ | —6 0 | —6 0 | —5 0 | —1 0 | —2 0 | —5 0 | 1 0 | 2 0 | 0 | 0 | 1 0 | 0 | 8 0 | 10 0 | 6 0 |
| | | Median $Q_2$ | —2 0 | 0 | 6 0 | 5 0 | 11 0 | 2 0 | 4 0 | 1 0 | 27 0 | 16 0 | 5 0 | 1 0 | 11 0 | 18 0 | 21 0 |
| | | $Q_3$ | 2 0 | 7 0 | 13 0 | 10 0 | 15 0 | 8 0 | 16 0 | 32 0 | 48 0 | 17 0 | 8 0 | 10 0 | 27 0 | 11 0 | 57 0 |
| 32/1 | ($N$ = 18) | Average | 3 2 | 3 7 | 11 2 | 18 2 | 22 0 | 4 3 | 8 1 | 13 9 | 15 6 | 13 1 | 7 5 | 12 1 | 25 1 | 33 8 | 35 1 |
| | | $Q_1$ | —3 0 | —4 0 | 1 0 | 2 0 | 9 0 | 0 | 2 0 | —2 0 | 2 0 | 0 | 2 0 | 5 0 | 5 0 | 11 0 | 12 0 |
| | | Median $Q_2$ | 1 0 | 1 0 | 13 0 | 21 0 | 21 0 | 5 0 | 5 0 | 5 0 | 6 0 | 7 0 | 5 0 | 6 0 | 20 0 | 27 0 | 20 0 |
| | | $Q_3$ | 10 0 | 15 0 | 18 0 | 31 0 | 33 0 | 6 0 | 12 0 | 21 0 | 25 0 | 20 0 | 10 0 | 16 0 | 27 0 | 60 0 | 57 0 |
| 74/1 | ($N$ = 6) | Average | 1 0 | —1 0 | 6 6 | 14 2 | 4 3 | 9 0 | 23 9 | 37 2 | 38 8 | 15 7 | 13 0 | 22 9 | 13 8 | 53 0 | 50 0 |
| | | $Q_1$ | —5 0 | —6 0 | —9 0 | —6 0 | —9 0 | —4 0 | 4 0 | 5 0 | 2 0 | 0 | —2 0 | —3 0 | 9 0 | 11 0 | —8 0 |
| | | Median $Q_2$ | 0 | —3 0 | 6 0 | 8 0 | —4 0 | 12 0 | 30 0 | 16 0 | 42 0 | 47 0 | 12 0 | 25 0 | 11 0 | 71 0 | 72 0 |
| | | $Q_3$ | 15 0 | 7 0 | 23 0 | 20 0 | 17 0 | 19 0 | 62 0 | 50 0 | 69 0 | 71 0 | 22 0 | 50 0 | 5 0 | 85 0 | 86 0 |
| 102/1 | ($N$ = 6) | Average | 0 6 | 0 4 | 6 3 | 11 0 | 23 8 | 13 9 | 31 4 | 31 0 | 38 7 | 27 8 | 11 5 | 31 8 | 10 3 | 52 7 | 51 6 |
| | | $Q_1$ | —2 0 | —3 0 | —7 0 | —3 0 | 0 | 3 0 | 2 0 | 3 0 | 19 0 | 1 0 | 0 | 7 0 | 6 0 | 11 0 | 5 0 |
| | | Median $Q_2$ | 0 | 2 0 | 10 0 | 12 0 | 7 0 | 13 0 | 15 0 | 6 0 | 31 0 | 6 0 | 16 0 | 17 0 | 50 0 | 19 0 | 60 0 |
| | | $Q_3$ | 3 0 | 9 0 | 15 0 | 28 0 | 52 0 | 23 0 | 51 0 | 56 0 | 55 0 | 18 0 | 26 0 | 56 0 | 84 0 | 81 0 | 91 0 |
| C/3 | ($N$ = 6) | Average | 3 0 | —0 3 | 1 1 | 11 2 | 7 0 | 2 8 | 3 8 | 8 8 | 7 3 | 4 8 | 5 8 | 3 5 | 13 2 | 18 5 | 11 8 |
| 13/3 | ($N$ = 3) | Average | 2 7 | 1 7 | 13 0 | 28 3 | —2 7 | 0 7 | 8 7 | 11 0 | —1 3 | 31 3 | 3 4 | 10 4 | 24 0 | 27 0 | 28 6 |
| 24/3 | ($N$ = 19) | Average | —0 9 | 2 8 | 4 1 | 6 4 | 13 6 | 3 6 | 4 4 | 6 4 | 6 7 | 4 6 | 2 7 | 7 2 | 10 5 | 13 1 | 18 2 |
| 30/3 | ($N$ = 7) | Average | —1 9 | 0 3 | 5 1 | 13 9 | 14 7 | 0 | 9 7 | 9 4 | 2 1 | 10 4 | —1 9 | 10 0 | 14 5 | 16 0 | 25 1 |
| *Weapon B* | | | | | | | | | | | | | | | | | |
| 15/1 | ($N$ = 16) | Average | 3 2 | —0 4 | 16 1 | 27 1 | 23 3 | 2 2 | 3 8 | 4 6 | —0 5 | 1 8 | 5 4 | 3 4 | 20 7 | 26 6 | 25 1 |
| 30/1 | ($N$ = 21) | Average | 1 7 | 2 3 | 6 7 | 13 1 | 23 4 | —0 2 | 2 3 | 1 5 | 4 6 | 1 5 | 1 5 | 4 6 | 11 2 | 17 7 | 27 9 |
| | | $Q_1$ | —1 0 | —3 0 | —1 0 | 0 | 7 0 | —3 0 | —1 0 | 1 0 | —4 0 | —2 0 | —2 0 | —1 0 | 1 0 | 4 0 | 6 0 |
| | | Median $Q_2$ | 0 | 1 0 | 5 0 | 9 0 | 19 0 | 1 0 | 1 0 | 1 0 | 3 0 | 3 0 | 1 0 | 1 0 | 6 0 | 10 0 | 21 0 |
| | | $Q_3$ | 5 0 | 8 0 | 17 0 | 21 0 | 40 0 | 3 0 | 4 0 | 8 0 | 12 0 | 10 0 | 6 0 | 9 0 | 24 0 | 24 0 | 55 0 |

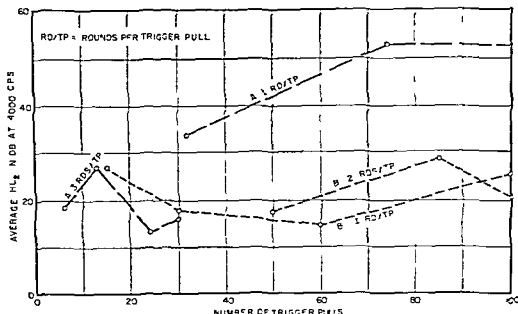



Fig 4 Showing average HL at 4000 cps following exposure to single and multiple rounds per trigger pull Parameter is weapon and rounds per trigger pull

## Effects of Firing Multiple Round Bursts

HL values taken at 4000 cps for single and multiple round firing conditions for certain weapons (see Table 1) are shown in Fig 4 It is interesting to note that the amount of HL would appear to be roughly the same for a given weapon and number of trigger pulls, regardless of whether 1 2, or 3 projectiles were fired To properly evaluate this result, we must consider the short interval of time between rounds—33 msec for the weapon A and 80 msec for weapon B

Two possible explanations for this finding are (1) the ear may be "refractory" and incapable of additional fatigue until a sufficient time has elapsed to allow for some recovery from the fatigue engendered by the first round, (2) the aural reflex activated by the first round of fire attenuates the transmission of the sound from the succeeding round or rounds of fire, so that no auditory fatigue is added to that caused by the first round This latter explanation is weakened somewhat by the fact that the time required for the full contraction of the reflex has been reported to be of the order of 100 or 150 msecs (22) In any event these data would suggest that a very rapid firing schedule of more than one round per second would apparently cause in the average ear a minimum amount of auditory fatigue or TTS for a given number of rounds fired

The five second interval between trigger pulls in the present study was chosen as being representative of the interval found during training ex-

TABLE 5 $Q_3$ *(75 per cent percentile) for $HL_2$ and peak SPL for the different weapons*

| Weapon | Peak SPL (dB re 0.0002 dynes/cm²) | No. trigger pulls | Test frequencies in cps: 500[a] | 1000 | 2000 | 3000 | 4000 | 6000 | Average at: 500 1000 2000 | 1000 2000 3000 |
|---|---|---|---|---|---|---|---|---|---|---|
| A | 172.5 | 102 | 11[a] | 26 | 56 | 84 | 84 | 91 | 31 | 55 |
| | | 74 | 7 | 22 | 50 | 78 | 85 | 86 | 26 | 50 |
| | | 32 | 0 | 10 | 16 | 27 | 60 | 57 | 9 | 18 |
| | | | | | | | Grand average | | 22 | 41 |
| B | 168.5 | 100 | 0 | 10 | 12 | 43 | 52 | 57 | 7 | 22 |
| | | 60 | 0 | 8 | 10 | 22 | 25 | 29 | 6 | 13 |
| | | 30 | 0 | 6 | 9 | 21 | 24 | 55 | 5 | 13 |
| | | | | | | | Grand average | | 6 | 16 |
| C | 167.5 | 97 | 0 | 7 | 10 | 12 | 14 | 27 | 6 | 10 |
| | | 63 | 0 | 11 | 12 | 38 | 55 | 56 | 7 | 20 |
| | | 23 | 0 | 12 | 18 | 51 | 65 | 73 | 10[b] | 27[b] |
| | | | | | | | Grand average | | 6 | 15 |
| D | 159.0 | 100 | 0 | 7 | 8 | 14 | 33 | 45 | 5 | 10 |

[a] $HL_2$ for 500 cps is estimated

[b] Only five ears involved in this firing condition and results are not included in grand average

ercises with these weapons when they are functioning properly. Inasmuch as the aural reflex normally relaxes within one second or so following exposure to an impulse of sound, the ear is presumably unprotected by the action of the aural reflex for the sound from successive rounds of fire that were separated by five seconds.

Reid (19) in an earlier study reports data on the effect of firing interval on TTS that is in general agreement with our results and assumptions.

## PHYSICAL MEASUREMENTS OF THE GUN NOISE

The acoustical evaluation of the gun noise included determining the peak sound-pressure level (SPL), and a time history and spectrum analysis of the pressure waveform. These data were obtained for the position where the firer's left ear would be and at a position 160 inches from the muzzle, at an azimuth of 255° from the line of fire.

### PRESSURE WAVEFORM

Physical measurements were made of the gun noise using a Ballistics Research Laboratories (BRL) 250KC shock-tube pressure transducer, connected to a Tektronix Type 531 oscilloscope which was photographed with

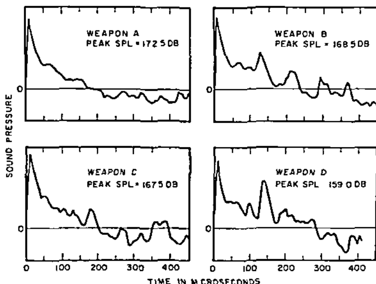



**Fig 5 Pressure waveforms and peak sound pressure level for the four types of weapons The peak levels of the waveforms have been adjusted to have approximately equal peak amplitude**

a Tektronix camera The transducer was calibrated the day of the measurement at 171 dB in the BRL shock-tube The oscilloscope was calibrated at the firing site with a standard cell Four oscillograms were photographed of the pressure wave produced by each weapon Figure 5 shows examples of the time history and peak sound-pressure level produced by the four weapons measured It is perhaps worth mentioning that the waveforms and peak pressures found for a given weapon were found to be extremely constant from one round of firing to the next

There is evidence which indicates that in addition to the peak pressure the duration of the pulse is important in predicting hearing impairment Examination of Fig 5 indicates that all four rifles have approximately the same duration, approximately 200 microseconds when defined as the time for the pressure wave to increase to and return from its initial positive peak to ambient When the duration is defined as the time required for the envelope of the pressure wave to decrease 20 dB below its peak, the duration is approximately 2 to 2 5 milliseconds

Measurements taken at the adjacent firer's head position (20 feet distant from the weapon being fired) revealed that the peak pressure produced by the rifles at this position were substantially less (8–12 dB) than at the firer's ear

## Spectrum Analysis

Spectral analyses were performed of the waveforms shown in Fig 5 To do this, appropriate "masks" of the waveforms were prepared for a

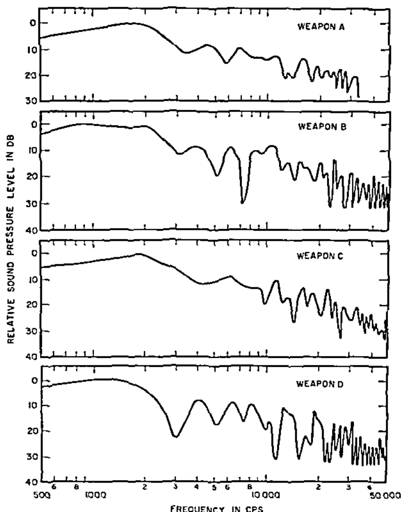



Fig 6 Showing spectrum level of impulses generated by the firing of weapons A B C and D

"pho oformer" * The line spectra were measured and analyzed as follows (a) the output of the photoformer was applied to a Hewlett-Packard Model 302 A Wave Analyzer, (b) a General Radio Type 908 P1 Synchronous Dial Drive was used to change the center frequency of the constant bandwidth analyzing filter at a rate of 7 cps/sec, (c) the amplitude spectra were recorded with a General Radio Type 1521-A Graphical Level Recorder, and (d) the envelope spectra were then replotted on a log frequency scale, omitting the 10 cps line structure produced by the 100 msec repetition period of the scanning photoformer These spectra are shown in Fig 6

The photoformer (3) is a photoelectric servo mechanism that observes an oscilloscope on which an opaque mask of a waveform to be analyzed is placed The photoformer was made to scan the mask 10 times per second and in so doing generate a train of impulses t be analyzed The envelope of the line spectrum of this impulse train is a close approximation to the true spectrum of the waveform we wish to analyze up to frequencies of pp oximately twenty times the reciprocal of the impulse duration

# CORRELATION BETWEEN SPECTRA, PEAK SOUND-PRESSURE LEVEL AND $HL_2$

## PEAK SPL AND $HL_2$

If the spectra shown in Fig 6 had been obtained from analyses of steady state stimuli, the maximum shift in threshold of hearing would be expected to occur at one octave above the stimulating frequency component having the highest sensation level (usually the frequency components having the highest SPL). Since the spectra of the impulses from these weapons appear to be broadly peaked at 2000 cps, the maximum auditory fatigue should occur at 4000 cps Our audiometric data (see Fig 2) in general would agree with this prediction

However, these spectra were obtained from analyses of the waveform of the impulses presumably present at the entrance to the ear canal Inasmuch as the natural behavior of the middle ear mechanical transmission system may interact with the temporal aspects of the waveform of one impulse somewhat differently than it does to those of another, the acoustic spectra of the impulses may be differentially changed during transmission through the middle ear structures, further knowledge of the transfer characteristics of the middle ear structures to brief impulses of sound is required before we can be certain that the apparent correlation betwen impulse spectra and frequency locus of auditory fatigue is real

The rifles tested are ranked in Table 5 according to peak sound pressure levels measured at the firer's head position For these particular weapons there is an apparently near perfect correlation between peak sound-pressure levels and damage risk to hearing consider, for example, the decreasing size of the grand averages of HL as the peak sound pressure levels from rifle to rifle decreased However because impulses from the weapons tested were similar in most respects (except peak sound pressure level), it does not follow that acoustic impulses having different rise and decay time characteristics as well as different peak SPL's will necessarily show the same high correlation we found between peak SPL and HL It is likely that other aspects besides peak pressure such as rise and decay time which, of course are related to spectrum content, also have some influence on auditory fatigue from acoustic impulses

## MAXIMUM TOBERABLE PEAK LEVELS

Our data are probably insufficient to define tolerable impulse-noise limits adequately even when certain rather far-reaching assumptions are made However, these data are, to our knowledge, the best available that bear directly on this problem, and might therefore be utilized for that purpose

In Fig 7 we have plotted, and extrapolated, some of our HL data as a

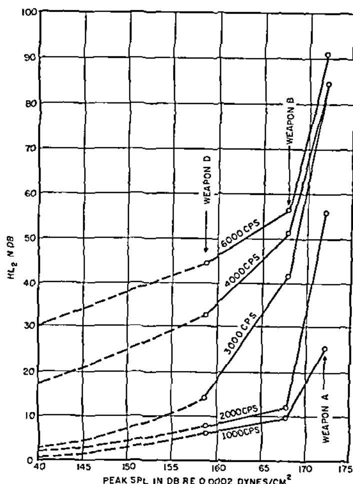



Fig 7 $HL_2$ for $Q_3$ as a function of peak SPL 100 trigger pulls one round per pull at the rate of one every 5 seconds Parameter is audiometric test frequency (see Table 5)

function of peak SPL of the impulses The curves in Fig 7 suggest that there are certain peak pressure levels above which the ear seems to be traumatically affected above this level threshold shifts increase at a tremendous rate for each one dB increase in peak SPL Although this level may be somewhat different for different audiometric test frequencies for these impulses the critical SPL appears to be about 168 dB for 25 percent of the ears A somewhat analogous finding was obtained by Miller Watson and Covell (14) in an experiment in which cats were exposed to intense noise These investigators found a critical sound pressure level and duration of exposure above which traumatic injury to cochlear structures occurred and below which only, primarily, temporary threshold shifts occurred

It is perhaps worth noting that the growth of the $HL_2$ as a function of peak SPL below this apparently critical level is similar to that observed with steady state noise (7, 10)

## COMPARISON OF HEARING-LOSS DATA WITH "DAMAGE-RISK CRITERIA"

It is not yet possible to predict with any confidence how many exposures to a given impulse-noise condition will produce a permanent hearing loss similar to the temporary loss suffered from a single exposure. The following considerations, however, do permit a guess that the HL levels found for the conditions studied in these tests could eventually become permanent

a It has been found with steady state noise that the amount of permanent hearing loss that will ensue after several years of work-day exposure to a given noise environment will be about equal to the amount of $TTS_2$ that one day's exposure will cause in the average normal ear. This relation appears to hold at least when the TTS values fall roughly in the average range of 10–30 dB (11)

b While it is, no doubt, unrealistic to expect a soldier to fire as often as required for these tests (a maximum of 100 trigger pulls) nearly every day for a number of years, he will also be exposed to some unknown degree to other severe noise conditions—when riding in a tank, firing other weapons, etc

If we tentatively accept, at least for present purposes, the propositions that $TTS_2$ eventually becomes a permanent threshold shift or hearing loss and that the HL values found in this study are reasonably representative of the $TTS_2$ values that would be found in normal young adult ears following the exposures conditions studied, then the HL values in Table 5 represent the approximate amount of permanent hearing loss that would be equaled or exceeded in 25 percent of the soldiers habitually firing a given weapon for various numbers of trigger pulls per day

In addition to the $Q_3$ values for the individual test frequencies, the average of the Q values for 500 (estimated), 1000, and 2000 cps, and the average Q values for 1000, 2000, and 3000 cps are given in Table 5 These averages are presented because they are being used (14), or have been suggested (12) for use as appropriate for evaluating the ability of a person to understand speech An average of 15 dB at these three frequencies has been proposed as the degree of hearing loss at which compensation for a handicap in hearing speech should begin

It must be made clear that the assumption that $TTS_2$ from impulse noise will eventually become a permanent threshold shift from repeated exposures is no more than a guess It is quite possible that the development of permanent hearing loss from impulse noise follows a much different pattern than that for permanent hearing loss from exposure to so-called steady-state noise In that regard, we are particularly impressed by the great variability of the threshold shifts exhibited by different ears as

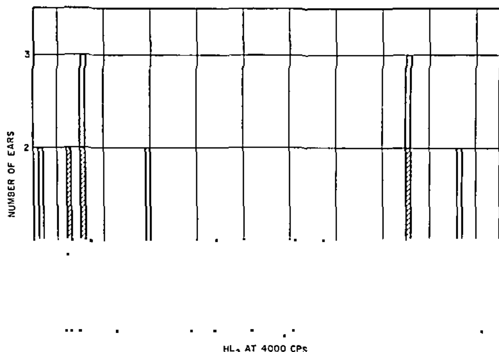



Fig 8 $HL_2$ at 4000 cps for the near ear (shaded area) and far ear (open area) following 100 single round trigger pulls with weapon B

the result of exposure to gun noise Indeed, it appears that the distribution of sensitivity to hearing loss from exposure to impulse noise is bimodal—*that there are "tender" ears and "tough" ears This bimodality is suggested* in the sample distributions of HL for weapon B as shown in Fig 8 Similar but not quite so obvious bimodal distributions were found with the data for the other test conditions as well

This bimodality, if indeed real, could, of course, be a reflection of invariant differences between "tough" and "tender" ears, or it could also be attributable to a sudden change in susceptibility within the same ear—that is, an ear may reach a "break point" up to which it is highly resistant and beyond which it tends to become suddenly and severely affected or traumatized

We have taken the liberty of estimating from Fig 7, and on the basis of the distributions of HL, we found in this study, as well as distributions of hearing loss found in studies of industrial noise as a cause of hearing loss (11), what the expected eventual permanent hearing level (ASA Standard (2)) might be in certain percentages of ears following repeated exposure to rifle noise of various peak SPL's at the listener's ears The results are given in Table 6 Caution in the use of Table 6 is in order in view of some of the extrapolation and rounding-off of data performed in its derivation

TABLE 6 *Estimated expected permanent hearing level (ASA Standard) to be equaled or exceeded in 50 %, 25 % and 10 % of ears following repeated exposure to about 100 rounds, at 5 sec intervals of the noise from shoulder rifles*

Peak SPL s are specified at the listener s ears

| | Test frequency in cps | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1000 | | | 2000 | | | 3000 | | | 4000 | | | 6000 | | |
| Peak SPL | 50 % | 25 % | 10 % | 50 % | 25 % | 10 % | 50 % | 25 % | 10 % | 50 % | 25 % | 10 % | 50 % | 25 % | 10 % |
| 170 dB | 0 | 15 | 25 | 10 | 25 | 35 | 35 | 55 | 70 | 45 | 65 | 85 | 50 | 70 | 90 |
| 165 | 0 | 9 | 16 | 0 | 10 | 20 | 12 | 32 | 42 | 25 | 45 | 60 | 47 | 52 | 67 |
| 160 | 0 | 7 | 15 | 0 | 8 | 16 | 0 | 18 | 25 | 15 | 35 | 45 | 25 | 45 | 60 |
| 150 | 0 | 3 | 10 | 0 | 4 | 15 | 0 | 8 | 15 | 10 | 25 | 35 | 20 | 40 | 50 |
| 140 | 0 | 0 | 0 | 0 | 2 | 5 | 0 | 2 | 10 | 5 | 18 | 30 | 10 | 30 | 45 |

## SUMMARY

Tests of the threshold of hearing of both ears of 178 soldiers before and after the firing of shoulder rifles at the rate of one trigger pull every five seconds indicate the following

a Significant hearing losses greater than 15 dB would probably occur in something over 10 % of the people repeatedly exposed to 100 acoustic (gun noise) impulses per day

1 at and above 4000 cps when the peak SPL exceeds 130–140 dB,
2 at and above 3000 cps when the peak SPL exceeds 150 dB
3 at and above 2000 cps when the peak SPL exceeds 160 dB,
4 at and above 1000 cps when the peak SPL exceeds 165 dB

b A criteria of acceptability for impulse noise having time-pattern characteristics similar to those of the gun noises of this study might be as follows

1 in terms of compensation standards based on the test frequencies of 500 1000 and 2000 cps 160 dB peak SPL
2 in terms of proposed standards for hearing impairment for speech (use of frequencies up to 3000 cps), 150 dB peak SPL,
3 in terms of good hearing for speech as well as other sounds, 140 dB peak SPL

## ACKNOWLEDGMENTS

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S U P P L E M E N T U M 212

# EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE BILDUNGSSTÄTTEN UND DEN STOFFAUSTAUSCH DER PERILYMPHE

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ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 212

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VON

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Diese Arbeit wurde durchgeführt mit Unterstützung der Deutschen Forschungsgemeinschaft sowie mit Kernforschungsmitteln des Landes Bayern und mit Mitteln der Gesellschaft der Freunde und Förderer der Universität München

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# INHALTSVERZEICHNIS

# I. EINLEITUNG UND PROBLEMSTELLUNG

Für die Physiologie des Hörens und die Pathogenese vieler Innenohrerkrankungen ist die Kenntnis über die Herkunft, den Stoffwechsel und die Strömung der Labyrinthflüssigkeiten von großer Bedeutung. Unsere Forschungsergebnisse darüber beruhen zum größten Teil auf dem Studium der Histologie und der pathologischen Anatomie, die in letzter Zeit besonders durch elektronenoptische und elektrophysiologische Untersuchungen, sowie durch klinische Arbeiten ergänzt worden sind.

Die Entdeckung von v. BÉKÉSY (1951, 1953), daß die Endolymphe der Scala media ein positives Gleichspannungspotential gegenüber der Perilymphe aufweist, regte auch die Erforschung der chemischen Verhältnisse im Innenohr und insbesondere in den Labyrinthflüssigkeiten an. Diese Untersuchungen stießen anfangs auf große Schwierigkeiten, da zunächst Methoden gefunden werden mußten, die zur Gewinnung der Labyrinthflüssigkeiten führten. Außerdem war es schwer, diese winzigen Mengen (Perilymphe pro Innenohr ca. 8–12 μl, Endolymphe ca. 2 μl) genau zu messen und chemisch zu untersuchen.

Neben rein chemisch-analytischen Versuchen hat das Studium der Sekretions- und Resorptionsstellen der Labyrinthflüssigkeiten viele Forscher beschäftigt, ohne daß eine endgültige Klärung erzielt werden konnte.

Bis vor einigen Jahren unterschied man beim Labyrinthliquor (KOBRAK 1949) zwei Flüssigkeiten: Die Peri- und die Endolymphe. Seit 1960 spricht man noch von einer Cortilymphe (ENGSTRÖM), und es wird damit jene Flüssigkeit bezeichnet, die den inneren und äußeren Tunnel, den NUEL'schen Raum und die Spalten zwischen den Haar- und Stützzellen durchströmt. Die Cortilymphe hat extrazellulären Charakter (RAUCH 1960) und ihre Herkunft aus der Perilymphe oder den HENSEN'schen Zellen (ENGSTRÖM 1960) wird diskutiert.

Bezüglich der Entstehung der Endolymphe nimmt man seit den Untersuchungen von SHAMBAUGH (1907, 1909), GUILD (1927), RÜEDI (1951) und den neueren autoradiographischen Versuchen von MEYER ZUM GOTTESBERGE (1960), PFISTER (1960) und KOBURG und PLESTER (1961) allgemein an, daß sie von der Stria vascularis produziert wird, während für die Resorption die Zellen des Sulcus spiralis externus (FRANDT und SAXÉN 1937), des Sacculus endolymphaticus (GUILD 1927, ALTMANN und WALTNER 1947, LUNDQUIST 1964) und der Stria vascularis (RÜEDI 1951, RAUCH 1962) verantwortlich gemacht werden.

Die Herkunft der Perilymphe ist noch völlig ungeklärt. Nach der bisherigen Meinung stammt die Perilymphe entweder aus dem Liquor cerebro-

spinalis (KARLEFORS 1923, KARBOWSKI 1930, MEURMANN 1931, QUIX und v. EGMONT 1933, PERLMANN und LINDSAY 1939, GISSELSON 1949, KLEY 1951, SVANE-KNUDSEN 1958, JAKO und Mitarb. 1959) oder aus der Endolymphe (WEBER LIEL 1879, QUIX und v. EGMONT 1933, WITTMAACK 1936, ALDRED, HALLPIKE und LEDOUX 1940, ALTMANN und WALTNER 1950, MYGIND 1952 und SVANE-KNUDSEN 1958) oder aus beiden Flüssigkeiten. Nachdem der Aquaeductus cochleae anatomisch ein Gebilde darstellt, das den Perilymphraum mit dem Subarachnoidalraum verbindet, hatte die Liquorgenese lange Zeit die meisten Anhänger. Gegen ein einfaches Liquordialysat der Perilymphe sprechen aber der unterschiedliche Eiweißgehalt dieser beiden Flüssigkeiten, sowie die elektrophoretischen Untersuchungen von CHEVANCE, GALLI und JEANMAIRE (1960). Auch die Zusammensetzung der Aminosäuren der Perilymphe verhält sich anders als die im Liquor (ANTONINI, CASORATI und CRIFO 1956/1957, CHEVANCE, GALLI und JEANMAIRE 1960, KLEYSKENS und RABAEY 1960). Diese Autoren, sowie GRAI und PORETTI (1951), welche erstmals Versuche mit radioaktiven Stoffen durchführten, waren der Anschauung, daß die Perilymphe auch ein Blutfiltrat darstellen könnte.

In unserer Klinik stehen Untersuchungen über die Ursprungsquellen und den Stoffwechsel der Perilymphe seit langem im Mittelpunkt des Interesses. Durch chemisch analytische und vor allem durch Experimente mit radioaktiven Stoffen haben wir versucht, zur Klärung der erwähnten Probleme beizutragen.

Wir stellten uns folgende Fragen: Ist die Perilymphe chemisch mit dem Liquor cerebro spinalis identisch? Ist der Aquaeductus cochleae für Flüssigkeiten durchgängig und, wenn ja, ist die Durchgängigkeit abhängig von der Molekulgröße? Ist die Reißner'sche Membran durchlässig für Jonen und größere Moleküle? Lassen sich Anhaltspunkte gewinnen, ob eine Produktion der Perilymphe im Perilymphraum selbst möglich ist und welche anatomischen Stellen kommen gegebenenfalls in Betracht? Welche Stoffwechselbeziehungen bestehen zwischen den einzelnen Labyrinthflüssigkeiten?

# II. EIGENE UNTERSUCHUNGEN

Zur Klärung unserer Probleme waren vor allem technische und methodische Fragen zu lösen. Sie stellen auch den Mittelpunkt unserer Darlegungen dar. Die methodischen Probleme bestanden vor allem darin, die physiologischen Verhältnisse im Innenohr, wie z. B. die ausbilancierten Druckverhältnisse, nicht zu stören. Eine weitere Schwierigkeit war, die Perilymphe ohne Blutbeimengungen zu gewinnen. Beide Probleme wurden durch die Gefriertechnik überwunden. Schließlich standen wir bei allen unseren Versuchen stets im Kampf mit dem Faktor „kleine Zahl", so daß selbst bei exaktester Durchführung der Meßmethoden Fehlerquellen von $\pm 5\%$ nicht auszuschliessen waren. Unter Berücksichtigung all dieser Schwierigkeiten war es deshalb erforderlich, die jeweilige Versuchsreihe an einer größeren Zahl von Tieren durchzuführen.

Unsere Untersuchungen wurden an 169 Kaninchen vorgenommen. Zur Auswahl dieser Tierspezies entschlossen wir uns deshalb, da die anatomischen Verhältnisse am Innenohr des Kaninchens neben dem des Affens dem Innenohr des Menschen am nächsten kommen. Außerdem gelingt es beim Kaninchen leicht, eine größere Menge Liquor zu gewinnen.

Die Tiere hatten ein Durchschnittsgewicht von ca. 4 kg. Es wurde darauf geachtet, daß die Kaninchen ohrgesund waren. Stellte sich bei den Experimenten ein krankhafter Befund am Ohr heraus, so wurde der Versuch nicht gewertet. Vor der Operation blieben die Tiere 12 Stunden nüchtern. Zur Narkose verwendeten wir 20%ige Urethanlösung, wovon wir pro kg Körpergewicht 7–8 ccm in die Ohrmuschelvene in zwei Raten langsam injizierten. Die Applikation der ersten Rate wurde beendet, sobald der PREYER'sche Ohrmuschelreflex vermindert war. Etwa 5 Minuten später erfolgte die Injektion der zweiten Rate. Die verabreichte Menge des Urethans war von Tier zu Tier verschieden. Die Narkose hielt gut über die durchschnittliche Operationsdauer von 3 Stunden an. Nach Beendigung des Versuches erhielten die Tiere, soweit sie noch gebraucht wurden, Depot-Penicillin und, wenn nötig, Flüssigkeitsersatzmittel.

### A) METHODEN

Für die Gewinnung der Labyrinthflüssigkeiten boten sich uns in der Literatur die Methode von ALDRED, HALLPIKE und LEDOUX (1940), GRAF und PORETTI (1950), sowie die Gefriertechnik nach RAUCH (1960) an. Diese Methoden wurden von den Autoren meist an Meerschweinchen ausgeführt und liessen sich nicht ohne weiteres auf das Kaninchen anwenden. Für

die von RAUCH (1960) beschriebene Kaltepräparation fehlte uns zudem noch die erforderliche aufwendige Ausstattung eines Kalteraumes. Aus diesem Grunde haben wir eigene Methoden entwickelt.

1. *Operative Methode zur Perilymphgewinnung*

Nach Enthaarung und Desinfektion im Operationsgebiet erfolgte in Urethannarkose nach lokaler Injektion von Novocain der Hautschnitt, der vom Vorderrand des Ohrmuschelansatzes senkrecht bis zur Mitte des absteigenden Unterkieferastes reichte. Nach Unterbindung der oberflächlichen Gefäße im Parotisbereich wurde der Wundsperrer eingesetzt und die Muskulatur sowie die Parotis freipräpariert. Der N. facialis wurde durchschnitten und die Bulla von der darüberliegenden Fascie freigelegt. Das weitere operative Vorgehen geschah unter der Operationslupe, bzw. dem Operationsmikroskop. Mit Hilfe eines Bohrers wurde die Bulla eröffnet und weite Teile des Bullaknochens wurden nach dorsal und ventral abgetragen. Dann wurde der knöcherne Teil des Gehörganges, der über die Gehörknöchelchen ragt, abgefräst. Ebenso wurde der Knochen zum Prozessus stylomastoideus zu entfernt, so daß man einen guten Einblick auf die knöcherne Schnecke, die Gehörknöchelchen, das ovale und runde Fenster gewinnen konnte (Abb. 1).

Bei all diesen Eingriffen wurden die Gehörknöchelchen geschont und es wurde sorgfältig darauf geachtet, daß der nicht luxierte Stapes in seiner Nische blieb. Ein grosses Gefäß, das quer durch die Bulla läuft, mußte unterbunden werden. Der N. facialis wurde aus seinem Kanal extrahiert. Hierbei kam es oft aus den Begleitgefässen zu Blutungen. Auch Liquorfluß konnte mitunter beobachtet werden. Beim weiteren Vorgehen wurden mit einem kleinen Bohrer zwei kleine Öffnungen in die knöcherne Schneckenwand gebohrt. Der Knochen wurde hierbei abgeschliffen und es gelang, ohne jede Blutung den Perilymphraum zu eröffnen. Die zweite Öffnung im Perilymphraum war zum Druckausgleich gedacht (Abb. 2). Der Endolymphschlauch leuchtete aus der Tiefe grau durch und bei der Entnahme der Perilymphe war darauf zu achten, daß er nicht verletzt wurde. Entscheidend war, daß die entnommene Perilymphe keine Blutbeimengungen enthielt. Wir haben zu diesem Zweck verschiedene Perilymphproben entweder auf dem Objektträger ausgestrichen und nach Erythrozyten gesucht oder wir wandten die Benzidinprobe an. Die Untersuchungen zeigten nur in den seltensten Fällen Blutbeimengungen. Bei Versuchen, die wir mit Jod-131-markierten Stoffen durchgeführt haben, wurde zuerst die Aktivität im Auffangglas gemessen und anschliessend untersucht, ob in der Perilymphe Blutbeimengungen enthalten waren. War dies der Fall, wurde der Versuch nicht gewertet.

2. *Beobachtungen bei der Perilymphgewinnung*

Wurde die Perilymphe mit einer Glaskapillare aus dem Perilymphraum im narkotisierten Tier entnommen, so war dieser Raum zunächst leer




Abb. 1. Mittelohr des Kaninchens (Operationssitus Stadium I). 1 Ovales Fenster mit Steigbügel. 2 Rundes Fenster mit Membran. 3 Stumpf des N. facialis. 4 Proz. styloides. 5 Knöcherne Gehörgangswand. 6 Trommelfellrest




Abb. 2. Mittelohr des Kaninchens (Operationssitus Stadium II). Das runde Fenster ist erweitert, die Fenstermembran ist zurückgeklappt, so daß die Einmündungsstelle des Aquaeductus cochleae (Pfeil) zu sehen ist

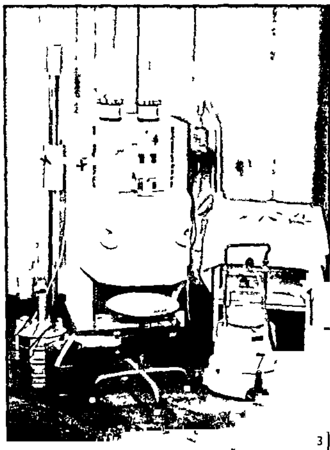



ABB. 3. Operationskryostat zur Mikropräparation des Innenohres und Gewinnung der Labyrinthflüssigkeiten (Beschreibung im Text).

Nach etwa 2–3 Minuten sah man, wie sich in der Skala von der Basalwindung her erneut Perilymphe nachbildete. Auch diese nachgelaufene Flüssigkeit haben wir gesammelt und, sobald diese entnommen war, konnte man das Nachlaufen noch zwei- bis dreimal beobachten. Wir haben die zuerst gewonnene Perilymphe als Fraktion I und die nachgelaufene Flüssigkeit als Fraktion II bezeichnet.

3. *Gefriermethode zur Gewinnung von Peri- und Endolymphe*

In Urethannarkose frästen wir nach Eröffnung der Bulla die knöcherne Schneckenwand und die Brücke zwischen rundem und ovalem Fenster bis auf eine dünne Schicht ab und dekapitierten anschließend das Kaninchen. Der Kopf fiel in flüssigen Stickstoff, bzw. in flüssiges Isopentan. Nach 2–3 Minuten wurde der Kaninchenkopf aus dem Kühlgefäß herausgenommen und in eine für diese Zwecke nach eigenen Angaben modifizierte Tiefkühltruhe (Kryostat Hersteller Fa. Dittes, Heidelberg) gebracht (Abb. 3). Das Kühlgerät, das mit Durchgriffen für die Manipulation und mit einem Fenster aus Thermopenscheiben versehen ist, ermöglichte die weitere Frei-

legung des Innenohres bei einer Temperatur von minus 18–22 Grad Der Kryostat ist innen beleuchtet und mit einem kleinen, höhenregulierbaren Operationstisch ausgestattet Die Instrumente wurden schon vorher in das Kuhlgerät gebracht, ebenso das Handstück der Bohrmaschine samt Anschluß nach außen Die Präparation des Innenohres geschah unter dem Zeiss-schen Operationsmikroskop, wie wir es ublicherweise auch in der Klinik bei mikrochirurgischen Eingriffen am Ohr verwenden Der Operationsabstand zwischen der untersten Thermopenplatte und Objekt betrug etwa 10 cm Unter 40-facher Vergrößerung wurde die restliche Knochenschicht der Labyrinthkapsel ganz abgefräst Perilymphe und der Endolymphschlauch lagen im gefrorenen Zustand vor Der Endolymphschlauch erschien etwas weißlich glänzend Es gelang zunächst, die gefrorene Perilymphe von Schneckenwindung zu Schneckenwindung zu entfernen und zu sammeln Der Endolymphschlauch wurde meist in Form kleiner Bruchstucke gewonnen Das kleine Sammelgefäß mußte zur Vermeidung von Verdunstung vorgekuhlt sein Die größte Schwierigkeit bei dieser Präparation bestand manchmal darin, daß es nicht gelang wegen der beengten Raumverhältnisse bestimmte Mikromanipulationen vorzunehmen Wir haben dann diese Eingriffe kurzfristig außerhalb des Kryostaten durchgeführt und wenn nötig den Kaninchenkopf später wieder neu eingefroren

4 *Methodik zur Endolymphgewinnung am lebenden Kaninchen*

Bei verschiedenen Versuchsanordnungen mit radioaktiven Stoffen konnten wir die Endolymphe nicht nach unserer Gefriermethode gewinnen, da das Tier am Leben bleiben sollte Außerdem hätte die Gefahr bestanden, daß die Membran des Endolymphschlauches, die außen mit radioaktiver Perilymphe in Beruhrung stand, ebenfalls radioaktiv war Diese Aktivität wäre also mitgemessen worden

Fur diese Versuche legten wir wieder den Perilymphraum durch 2 Bohrlöcher in der Schnecke frei und punktierten den Endolymphschlauch durch eine hauchdünne Kapillare an Selbstverständlich mußte darauf geachtet werden, daß das Operationsfeld blutfrei und die Perilymphe von der Oberfläche her abgesaugt war Bei radioaktiven Versuchen haben wir den Perilymphraum vorher mit Aqua destillata oder mit physiologischer Kochsalzlösung gespult und diese Spulflussigkeit vom Rande des Bohrloches wieder abgesaugt Durch die Kapillarität gelang die Aspiration von Endolymphe Der Endolymphschlauch war oft von unterschiedlicher Elastizität und setzte der Berührung mit der Glaskapillare mitunter großen Widerstand entgegen Die gewonnene Menge Endolymphe war nur sehr gering und analytisch kaum meßbar Sie reichte jedoch aus, um eine Aussage über eine meßbare Radioaktivität machen zu können

5 *Methodik zum Verschluß des Aquaeductus cochleae*

Nach Freilegung der knöchernen Schnecke nach der oben beschriebenen Methode war hier noch ein zusatzliches Abfräsen des nach dorsal reichen-

Tabelle 2 *Prozentuale Verteilung der Aminosäuren in Perilymphe (Fraktion I und II), Liquor und Serum*

Angaben in % und Integratormengeneinheiten (E)

| Substanz | Perilymphe Fraktion I E | % | Perilymphe Fraktion II E | % | Liquor E | % | Serum E | % |
|---|---|---|---|---|---|---|---|---|
| Histiden | 25 | 3,57 | 18 | 4,73 | 22 | 2,78 | 33 | 4,71 |
| Lysin | 47 | 6,71 | 28 | 7,37 | 48 | 6,08 | 74 | 10,57 |
| Arginin | 38 | 5,43 | 23 | 6,05 | 60 | 7,59 | 24 | 3,43 |
| Nicht ident. Substanzen | 144 | 20,57 | 96 | 25,26 | 166 | 21,01 | 75 | 10,71 |
| Asparagin Glyzin | 209 | 29,85 | 100 | 26,32 | 215 | 27,21 | 257 | 36,71 |
| Glutaminsäure Theorin | 100 | 14,28 | 55 | 14,46 | 143 | 18,10 | 95 | 13,57 |
| Alanin | 143 | 20,43 | 60 | 15,78 | 140 | 17,72 | 145 | 20,71 |
| Gesamtgehalt in Integratormengeneinheiten | 706 | | 380 | | 794 | | 703 | |

3 *Vergleichende Elektrophoresen von Perilymphe, Liquor und Blutserum*

Die Versuche wurden an 18 Kaninchen durchgeführt Die Serumelektrophoresen wurden nach Grassmann und Hannig (1958) angestellt Der Eiweißgehalt des Serums konnte refraktometrisch ermittelt werden Den Liquor gewannen wir durch Zisternenpunktion, während die Perilymphe teils nach der Gefriermethodik, teils nach der operativen Methode gewonnen wurde Erythrozytenhaltiger Liquor oder bluthaltige Perilymphe wurden nicht mituntersucht Den Gesamteiweißwert im Liquor und in der Perilymphe haben wir nach Fuhr (1955) gemessen Zur Elektrophorese wurde der Liquor und die Perilymphe nach Mies (1953) eingeengt und auf Acetatstreifen aufgetragen

Es zeigten sich hierbei folgende Ergebnisse (Tabelle 3)

Vergleicht man die Ergebnisse der einzelnen Körperflüssigkeiten, so fällt zunächst der 4–5 fache höhere Eiweißwert der Perilymphe gegenüber dem Liquor auf Der Unterschied hinsichtlich der Zusammensetzung der Globuline ist nur gering

## D) Untersuchungen mit radioaktiven Stoffen

Für unsere Untersuchungen über die Herkunft, die Bildungsstätten und den Stoffwechsel der Perilymphe hielten wir die Verwendung von radioaktiven Isotopen am besten geeignet Es ist bekannt, daß durch diese Stoffe die physiologische Zusammensetzung der Labyrinthflüssigkeiten, ihre Konzentration an Elektrolyten und andere biologische Faktoren, die bei diesen Untersuchungen berücksichtigt werden müssen, nicht gestört werden Außerdem haben sie den wesentlichen Vorteil gegenüber anderen Untersuchungsmethoden, daß auch kleinste Mengen gemessen werden können

Tabelle 3 *Eiweißfraktionen des Serums, des Liquors und der Perilymphe*

| | | |
|---|---|---|
| *Serum* | Albumin | 56,8 rel % |
| | $\alpha_1$ Globulin | 6,7 |
| | $\alpha_2$ Globulin | 6,6 |
| | $\beta$ Globulin | 16,1 |
| | $\gamma$ Globulin | 14,8 |
| Der Gesamteiweißwert des Serums betrug 5,7 g% | | |
| *Liquor* | Albumin | 53,0 rel % |
| | $\alpha_1$ Globulin | 4,5 |
| | $\alpha_2$ Globulin | 6,4 |
| | $\beta$ Globulin | 24,9 |
| | $\gamma$ Globulin | 11,1 |
| Der Gesamteiweißwert des Liquors betrug 21 mg% | | |
| *Perilymphe* | Albumin | 58,4 rel % |
| | $\alpha_1$ Globulin | 8,3 |
| | $\beta$ Globulin | 21,3 |
| | $\gamma$ Globulin | 11,5 |
| Der Gesamteiweißwert der Perilymphe betrug 96 mg% | | |

Wir verwendeten Phosphor-32 in wässeriger Lösung, dann verschieden markierte Aminosäuren, sowie Jod 131 markiertes Humanalbumin und kanincheneigenes Albumin und schließlich radioaktiv markierte Zellgranula.

Phosphor-32 findet für Traceruntersuchungen am meisten Verwendung, da er eine günstige Halbwertszeit von etwa 14 Tagen hat und seine Strahlung sich gut messen läßt. Der Nachteil ist, daß anorganisches Phosphat in relativ hohem Maße organisch gebunden wird, so daß beim Nachweis der Radioaktivität nicht ohne weiteres zu entscheiden ist, ob diese Aktivität durch Membranpassage oder durch die Mitwirkung von Zellen in den zu untersuchenden Raum gelangt ist.

In einer zweiten Versuchsreihe ergänzten wir deshalb unsere Untersuchungen mit Jod-131-markierten Albuminen, die sicher extrazellulär bleiben.

In einer anderen Versuchsreihe führten wir autoradiographische Untersuchungen mit tritiummarkierten Aminosäuren durch. Hierdurch gelang es, den Eiweißstoffwechsel in den Zellen und Gewebsarten der Schnecke und des Vorhofbogengangapparates in Abhängigkeit von der Zeit zu untersuchen. Wir hofften auf diese Weise auch Hinweise über den Antransport der Aminosäuren zu den Geweben des Innenohres erhalten zu können.

1. *Versuche mit radioaktivem Phosphor*

Wir verwendeten Phosphat 32 als Orthophosphat in verdünnter physiologischer Kochsalzlösung, sterilisiert und trägerfrei. Die spezifische Aktivität betrug 1 mc/g Phosphat. Die Substanz wurde intravenös, subokzipital und in den Perilymphraum selbst appliziert.

Die *Messung* der Aktivität beim Phosphor-32 erfolgte im Geiger-Müller-Zählrohr mit Endglimmerfenster (FRIESEKE und HOEPFNER FH 781, Fensterdicke 3,61 mg/ccm) bei einer Spannung von 750 Volt ohne Untersetzung mit Berger-Zeit Stoppuhr.

Die Voraussetzung für diese Art der Messung war, daß die zu messenden Körperflüssigkeiten mit einer gleichen Schichtdicke und in stets der gleichen räumlichen Anordnung auf das Meßschälchen aufgetragen wurden, so daß für die Messung stets gleiche Bedingungen gegeben waren. Die gewonnenen Flüssigkeiten auf dem Meßschälchen wurden eingedampft. Als Bezugsmenge wurde 25 $\mu$l der jeweils zu untersuchenden Körperflüssigkeit zugrunde gelegt. Da pro Innenohr nur 8–12 $\mu$l Perilymphe zu gewinnen waren, wurde die Aktivität dieser Menge in Beziehung gesetzt zu der Radioaktivität von 25 $\mu$l anderer Körperflüssigkeiten wie Liquor und Blut. Der statistische Umrechnungsfehler lag zwischen 4 und 5%. Bei der Messung selbst erfolgte zunächst die Bestimmung des Leerwertes an einem nicht aktiven Meßschälchen, dann wurde die Messung des aktiven Meßschälchens vorgenommen und hiervon der Leerwert wieder abgezogen. Unsere Werte geben also die absolute Impulszahlen bezogen auf eine Meßzeit von 10 Minuten wieder. Vielfach wurden die Proben in Anbetracht ihrer kleinen Mengen und der geringen Aktivität mehrmals gemessen. Zum Teil erfolgte eine Messung über 1 Stunde und das Ergebnis wurde in Beziehung gebracht zum 10-Minutenwert. Da die Meßbedingungen identisch gehalten wurden, erübrigten sich Korrekturen des Meßergebnisses bezüglich Raumwinkel, Reflexion an der Unterlage, Eigenabsorption und Eigenstreuung, Absorption in der Wand bzw. im Fenster des Zählrohres und in der Luftschicht zwischen Quelle und Zählrohr.

Bei den Traceruntersuchungen wurden folgende Versuchsanordnungen getroffen:

a) Bei 32 Kaninchen injizierten wir ca. 400 $\mu$C Phosphor-32, gelöst in 2 ccm Kochsalzlösung, in die Ohrlöffelvene. Nach 10 Minuten wurde, soweit die Versuchsdauer länger als 10 Minuten betrug, Blut aus der Vene des anderen Ohrlöffels als Bezugswert entnommen und mit einer Mikropipette 25 $\mu$l auf ein Meßschälchen aufgetragen. Die Tiere wurden in Abständen von 5 Minuten bis zu 168 Stunden nach der Injektion durch Evipan getötet. Anschließend eröffneten wir das Herz, damit das Tier ausblutete. Gleiche Mengen von Blut aus dem Herzen (25 $\mu$l) wurden auf ein Meßschälchen aufgetragen und gleichmäßig verteilt. Anschließend punktierten wir die Zisterna magna, gewannen den Liquor und trugen eine gleiche Menge auf ein Meßschälchen auf. Die Perilymphe wurde nach einer der zwei angegebenen Methoden gewonnen, mit Mikropipetten gemessen und auf das Meßschälchen aufgetragen und verrührt. Durchschnittlich gewannen wir 12 $\mu$l. Bei der Messung der Radioaktivität wurde diese kleine Menge in Beziehung gesetzt zu den 25 $\mu$l der anderen Körperflüssigkeiten und dann [illegible]rechend umgerechnet.

B[illegible] 12 Kaninchen injizierten wir das radioaktiv markierte Phosphar

suboccipital. Wir legten zu diesem Zweck die Membrana atlanto-occipitalis operativ frei und punktierten den subarachnoidalen Liquorraum. Es wurde zunächst etwa 0,5 ccm Liquor abgesaugt, die Nadel stecken gelassen, anschließend applizierten wir ohne Druck ca. 100 $\mu$C Phosphor 32, das in 0,5 ccm physiologischer Kochsalzlösung gelöst war. Die Radioaktivität wurde 5 Minuten bis 24 Stunden danach in der Perilymphe, im Liquor und im Blut gemessen.

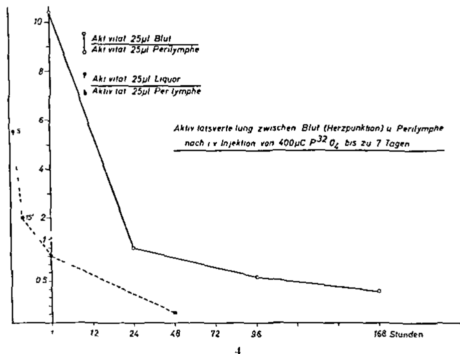
Bei den Versuchen zeigte sich, daß die innerhalb eines gleichen Zeitraums durchgeführten Untersuchungen größere Schwankungen hinsichtlich der Konzentration des Phosphors im Liquor und in der Perilymphe aufwiesen, obwohl stets gleiche Mengen an aktiver Substanz appliziert wurden. Es spielen offenbar physiologische Faktoren hinsichtlich der Ausscheidung, der peripheren Durchblutung und der Permeabilität der Bluthirnschranke eine große Rolle. Wir konnten deshalb keinen der meßbaren Aktivitätswerte als Bezugssystem nehmen und haben zur Veranschaulichung der Verteilungsverhältnisse nur die Quotienten aus der Aktivität des gleichen Blutvolumens zur Aktivität des entsprechenden Perilymphvolumens herangezogen. Die im Diagramm angeführten Werte sind Mittelwerte (Abb. 4 und 5).

Die ersten meßbaren Aktivitäten in der Perilymphe waren 5 Minuten nach der Injektion nachzuweisen. Der Quotient aus Blut und Perilymphe liegt bei 63, d. h., daß zu diesem Zeitpunkt das Blut noch 60mal aktiver ist als die Perilymphe. Nach 15 Minuten ist der Quotient auf 15 abgesunken. In der Folgezeit fällt die Aktivität weiter ab und nach 1 Stunde ist die Aktivität nur noch etwa 2mal höher als in der Perilymphe, während sie nach 24 Stunden ausgeglichen ist. Der Quotient des Liquorwertes zum Perilymphwert fällt ebenfalls konstant ab. Nach 15 Minuten sind die Aktivitäten praktisch ausgeglichen und nach 1 Stunde ist die Aktivität in der Perilymphe höher als die entsprechende Aktivität im Liquor.

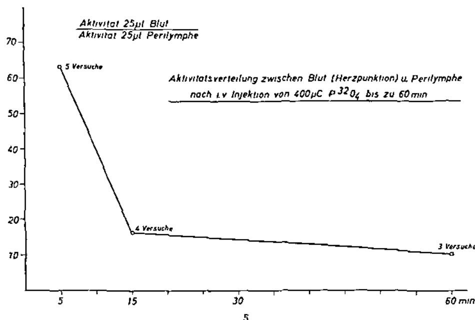
Zur Klärung der entscheidenden Frage, ob das aktive Phosphat über das Kapillarsystem oder auf dem Diffusionsweg über den Hirnliquor in die Perilymphe gelangt, injizierten wir Phosphor 32 nach der von uns angegebenen Methode suboccipital. Hierbei ergibt sich, daß nach 5 Minuten über 30 % der Phosphoraktivität des Liquors in der Perilymphe vorhanden war. Nach 15 Minuten betrug die Aktivität in der Perilymphe über 50 % der Aktivität des Liquors, sehr bald kam es dann zu einem Aktivitätsausgleich, der bis zu 24 Stunden keinerlei Verschiebung mehr zeigte.

Die Tabelle 4 gibt eine Übersicht der Aktivitätsverteilung nach intravenöser und suboccipitaler Applikation von Phosphor 32 (Mittelwert von je 5 Versuchen) wieder.

Aus dem Vergleich der suboccipitalen und intravenösen Applikation von Phosphor 32 ergibt sich, daß in Anbetracht der hohen Radioaktivität in der Perilymphe nach kurzen Zeitabständen der Phosphor sowohl über den Blutweg als auch über den Aquaeductus cochleae in den Perilymphraum gelangen kann.




4




5

ABB. 4 und 5. Aktivitätsverteilung zwischen Blut, Liquor und Perilymphe nach intravenöser Applikation von 400 µC $P^{32}O_4$ von 5 Minuten bis zu 7 Tagen (Mittelwerte)

c) Bei 17 Kaninchen applizierten wir ca. 100 µC-Phosphor-32 in einem Tropfen physiologischer Kochsalzlösung durch das operativ eröffnete ovale Fenster ohne Druck in das Vestibulum der einen Seite. Auf sorgfältigste Blutstillung vor der Applikation wurde geachtet und der umliegende Knochen mit Bienenwachs verkittet. Zur besseren Applikation wurde ein

Tabelle 4 *Vergleich der Aktivitätsverteilung nach intravenöser und subocccipitaler Applikation von Phosphor-32 (Mittelwerte), gemessen in Impulsen pro 25 μl Körperflüssigkeit*

Meßzeit 10 Minuten

| Applikation | Zeit | Blut | Liquor | Perilymphe |
|---|---|---|---|---|
| *Intravenös* | 5 Min | 7 222 | 399 | 112 |
| | 15 Min | 7 165 | 577 | 445 |
| | 60 Min | 1 951 | 1 014 | 1 135 |
| Subocccipital | 5 Min | 257 | 105 896 | 26 733 |
| | 15 Min | 588 | 75 547 | 39 865 |
| | 60 Min | 514 | 40 681 | 41 142 |

Schacht aus Bienenwachs in der rechten Bulla angelegt, der bis ins ovale Fenster reichte und durch eine Wachsplombe unmittelbar nach der Applikation von oben verschlossen wurde 1 Stunde nach Verabreichung der aktiven Substanz töteten wir die Tiere und gewannen neben Blut, Blutserum und Liquor die Perilymphe aus dem Ohr der anderen Seite

Außer Phosphor-32 applizierten wir bei dieser Versuchsanordnung auch noch die Aminosäure Glyzin in $C^{14}$-markierter Form, sowie Jod[131] markiertes Humanalbumin und Phosphor markierte Mitochondrien

Es zeigten sich hierbei folgende Ergebnisse Nach einer Versuchsdauer von 10 Minuten waren bereits geringe Radioaktivitäten im Blut nachweisbar Bei den Phosphor-Versuchen fanden sich frühestens nach 1 Stunde die ersten Aktivitäten im Liquor Die Radioaktivität lag hier größenordnungsmäßig so hoch wie die Radioaktivität im Blutserum Bei der Messung der Radioaktivität in der Perilymphe des anderen Ohres ergab sich, daß von den 17 Phosphorversuchen 14 mal die Aktivität hier höher war als die Aktivität der entsprechenden Menge des Liquors und nur in 3 Fällen lagen die Aktivitäten im Liquorvolumen höher als in der Perilymphe Die hier gefundenen Ergebnisse wiesen aber eine beträchtliche Streubreite auf, sodaß erst eine größere Versuchsreihe noch Aufschluß über diese Befunde geben könnte.

Glyzin in $C^{14}$-markierter Form direkt in den Perilymphraum gebracht, ließ sich in der Perilymphe des anderen Ohres noch in höherer Konzentration als im Hirnliquor bei der Messung am Methandurchflußzähler nachweisen

Bei der Applikation von phosphormarkierten Mitochondrien in den Perilymphraum fanden sich in der Perilymphe des anderen Ohres nach 1 Stunde Aktivitätswerte, die unter der Aktivität des Hirnliquors lagen Wahrscheinlich ging diese Aktivität auf beigemengtes Phosphat zurück, das in anorganisch freier Form vorlag

Wurden Jod[131]-markierte Albumine in den Perilymphraum gebracht, so

Tabelle 5. *Vergleich der Aktivitätskonzentration von Phosphor-32 in der Perilymphe bei verschlossenem und offenem Aquaeductus cochleae bei suboccipitaler Applikation*

(Impulse/25 $\mu$l Körperflüssigkeit. Meßzeit: 10 Minuten. Mittelwerte)

| Versuchs Nr. | Versuchs Dauer | Liquor | Perilymphe li. (Aq. c. verschlossen) | Perilymphe re. (Aq. c. offen) |
|---|---|---|---|---|
| 1 | 5 Min. | 30 782 | 738 | 11 781 |
| 2 | 5 Min. | 41 361 | 397 | 9 476 |
| 3 | 5 Min. | 37 981 | 401 | 10 512 |
| 4 | 5 Min. | 32 671 | 483 | 8 693 |

Die Zahlen geben die Impulse pro 10 Minuten Meßzeit bezogen auf 25 $\mu$l Körperflüssigkeit wieder.

war nach 1 Stunde noch keine Radioaktivität in der Perilymphe des anderen Ohres nachzuweisen, jedoch fand sich im Liquor eine Aktivität.

d) In 4 Versuchen plombierten wir den Aquaeductus cochleae der einen Seite und injizierten Phosphor-32 suboccipital. Nach ca. 5 Minuten töteten wir das Tier und entnahmen von der operierten Seite die inzwischen nachgeflossene Perilymphe und verglichen ihre Aktivität mit der gleichen Menge gewonnener Perilymphe der anderen bereits voroperierten Seite.

Die Tabelle 5 gibt die Ergebnisse wieder.

Aus der Tabelle ist ersichtlich, daß 5 Minuten nach suboccipitaler Applikation von Phosphor-32 eine hohe Aktivität in der Perilymphe des nichtoperierten Ohres und eine niedrige Aktivität in der inzwischen nachgelaufenen Perilymphe auf der Seite mit plombiertem Aquaeductus cochleae zu finden ist. Auf welche Weise bei letzterer die Aktivität vom Subarachnoidalraum in die Perilymphe gelangte, ist nicht sicher zu klären; möglicherweise geschieht der Eintritt des Phosphats in den Perilymphraum entlang der perineuralen Lymphscheiden des inneren Gehörganges.

Bei der suboccipitalen Applikation von organisch gebundenem Phosphat an Mitochondrien war auch nach 1 Stunde praktisch keine Aktivität in einem der beiden Perilymphräume nachzuweisen.

e) Zur Untersuchung der Durchgängigkeit der Reißnerschen Membran applizierten wir Phosphor-32 in den Perilymphraum selbst und gewannen anschließend durch Punktion des Ductus cochlearis mit einer Glaskapillare etwas Endolymphe. Vorher wurde die aktive Lösung im Perilymphraum wieder abgesaugt und letzterer mit Aqua destillata durchspült. Die kleine Menge Endolymphe war quantitativ kaum meßbar, weshalb wir die Spitze der Glaskapillare in einem Meßschälchen zerdruckten, um die Aktivität bei entsprechend langer Meßdauer bestimmen und mit einer ebenfalls durch Gl[illegible]llare gewonnenen Durchspülflüssigkeit aus dem Perilymphraum ver[illegible] zu können.

Bei der Auswertung der gewonnenen Befunde waren die Aktivitäten sehr gering. Es läßt sich lediglich aussagen, daß in der Endolymphe eine höhere Konzentration vorhanden war als in der gleichgroßen Menge gemessener Spülflüssigkeit aus dem Perilymphraum.

Wurde Thymin in $C^{14}$-markierter Form in eben derselben Weise appliziert, so waren die gleichen Versuchsergebnisse wie bei der Applikation von Phosphor-32 zu erzielen. Die Reißnersche Membran ist offenbar durchgängig für Ionen und kleine Moleküle in der Größenordnung kleiner Aminosäuren.

## 2. *Versuche mit Jod¹³¹ markierten Albuminen*

In einer weiteren Versuchsreihe führten wir in praktisch denselben Versuchsanordnungen wie bei den Phosphorversuchen unsere Untersuchungen mit $Jod^{131}$-markiertem Albumin durch. Dieses Isotop bleibt sicher extrazellulär. Es hat außerdem eine wesentlich höhere Molekulgroße (etwa 60000) als Phosphor-32, so daß sich in Verbindung mit den Phosphorversuchen auch Aussagen uber Diffusionsvorgange an den Membranen in Abhängigkeit von der Molekülgröße machen lassen.

Wir verwendeten $Jod^{131}$-markiertes Humanserumalbumin und selbst hergestelltes kanincheneigenes markiertes Serumalbumin.

Die von der Industrie gelieferten jodmarkierten Humanalbuminfraktionen erwiesen sich bei der Prüfung in der Tiselius-Apparatur und auch papierelektrophoretisch als frei von sicher erkennbaren Unreinheiten. In der Immuno-Elektrophorese ergab die Testung einen Reinheitsgrad von 98%.

Die Markierung der kanincheneigenen Albumine, die in dankenswerter Weise von Frau SCHEID-SEYDEL von der Nervenklinik der Universität München vorgenommen wurde, erfolgte nach der Methode von PRESSMANN und EISEN (1950). Die spezifische Aktivität unserer eigenen Praparation betrug 1–4 C/mg Albumin.

Die selbst hergestellten kanincheneigenen $Jod^{131}$ markierten Albumine und die bezogenen jodmarkierten Human-Albuminlosungen wurden gegen mehrfach gewechselte physiologische Kochsalzlosungen 48 Stunden dialysiert und anschließend durch eine Ionen-Austauschsäule (Amberlite IRA 400 OH) geschickt. Zur Sterilisierung filtrierten wir die $Jod^{131}$-Albuminlösung durch eine Glasfritte G 5/3 und es wurde die übliche bakteriologische Prufung auf Keimfreiheit angestellt. Die endgültige Ausbeute bei den selbst hergestellten Albuminlösungen betrug etwa 70% der Ausgangsalbuminlosung und 1,0–1,5% der ursprünglichen Aktivität. Mit Hilfe der papierelektrophoretischen Trennung konnte nachgewiesen werden, daß weniger als 0,5% in Form von freiem Jod vorliegen.

Nach der Methode von KUNKEL und WARD (1950) pruften wir das jodierte Albumin auf Reinheit der Praparation und auf Denaturierung. Die angelegten Trubungskurven mit $Jod^{131}$-Albumin und nativem Albumin stimmten

Tabelle 6. *Vergleich der Aktivitätskonzentration in Serum, Liquor und Perilymphe (Fraktion I und II) nach intravenöser Applikation von Jod*[131]*-markiertem Human-Albumin (Mittelwerte)*

| Zahl d. Fälle | Vers.-Dauer | Aktivität (= Impulszahl/25 $\mu l$) in % des Ausgangswertes | | | |
|---|---|---|---|---|---|
| | | Serum | Liquor | Perilymphe Fraktion I | Perilymphe Fraktion II |
| 5 | 10 Min | 100 | 0,11 | 0,13 | 0,15 |
| 6 | 60 Min | 72 | 0,13 | 0,22 | 0,23 |
| 7 | 220 Min | 68 | 0,15 | 0,20 | 0,29 |
| 8 | 48 Std | 20 | 0,16 | 0,31 | 0,13 |

überein und die methodische Fehlerbreite von $\pm 3\%$ wurde nicht überschritten.

Die *Messungen* der Aktivitäten führten wir mit dem Strahlungsmeßgerät FH 90 — Szintillationszähler mit Bohrlochkristall FH 451 durch.

Folgende Versuche wurden vorgenommen:

a) 22 Kaninchen erhielten je 100 $\mu$C Humanalbumin und 3 Kaninchen kanincheneigenes jodmarkiertes Albumin in die Ohrvene injiziert. Die Versuchsdauer betrug 10 Minuten bis 48 Stunden. Nach 10 Minuten wurde aus der Vene des anderen Ohres ein Tropfen Blut und Blut für die Serumgewinnung als Bezugswert abgenommen.

Zur Perilymphgewinnung präparierten wir am narkotisierten Tier das Innenohr nach der angegebenen Methode und entnahmen die beiden Fraktionen, die in je 1 Meßschälchen gegeben wurden. Anschließend punktierten wir die Zisterna magna zur Liquorgewinnung. Schließlich töteten wir das Tier und entnahmen aus dem Herzen Blut zur Direktmessung und Blut zur Serumgewinnung. Gleiche Mengen von den einzelnen Körperflüssigkeiten wurden jeweils in 1 Meßschälchen abpipettiert.

In der Tabelle 6 sind die Ergebnisse dieser Versuchsanordnung aufgezeichnet.

Die Aktivität ist diesmal ausgedrückt in Quotienten aus Impulszahlen pro 25 $\mu l$ untersuchte Körperflüssigkeit, bezogen auf den 10 Minuten Ausgangswert des Serums.

Eine Aktivität war frühestens nach 10 Minuten in der Perilymphe nachweisbar. Die Aktivität des Liquors lag zu diesem Zeitpunkt noch wesentlich unter der Aktivität der Fraktion I der Perilymphe, während die Aktivitätskonzentration in der Fraktion II nur etwas höher war als die Konzentration in der gleichen Menge Liquor. Nach 60 Minuten verringerte sich der Abstand zwischen der Konzentration im Liquor und in der Perilymphe. D[illegible]ivität in der Perilymphe war nur noch doppelt so groß wie im Liq[illegible]h war die Konzentration in der nachgeflossenen Perilymphe F[illegible] II) [illegible]her als in der Fraktion I. Nach 2 Stunden war das Ergebnis

TABELLE 7. *Vergleich der Aktivitätskonzentration in Serum, Liquor und Perilymphe (Fraktion I und II) nach Applikation von kanincheneigenen Albuminen (Mittelwerte).*

| Zahl d Fälle | Vers Dauer | Aktivität (= Impulszahl/mg Gesamteiweiß) in % des Ausgangswertes | | | |
|---|---|---|---|---|---|
| | | Serum | Liquor | Perilymphe Fraktion I | Perilymphe Fraktion II |
| 1 | 10 Min | 100 | 3,7 | 30,5 | 8,4 |
| 1 | 60 Min | — | 7,7 | 25,9 | 19,5 |
| 1 | 120 Min | 92 | 14,9 | 70,0 | — |

noch deutlicher 48 Stunden nach Applikation von markiertem Albumin war die Aktivität im Serum nur noch 20% des Ausgangswertes und die Perilymphkonzentration in Fraktion I war wieder am höchsten

Um den Einwand zu entkräften, daß das körperfremde Humanalbumin die Ergebnisse beeinträchtigt, führten wir dieselben Versuche mit kanincheneigenem jodmarkierten Albumin durch Bei der Auswertung dieser 3 Versuche haben wir gleichzeitig das Gesamteiweiß im Serum, Liquor und in beiden Perilymphfraktionen bestimmt

In der Tabelle 7 sind unsere Versuche mit kanincheneigenem jodmarkierten Albumin wiedergegeben

Die Radioaktivität wurde diesmal ausgedrückt im Quotienten aus der Impulszahl zur Gesamteiweißmenge der jeweiligen Körperflüssigkeit und in Beziehung gesetzt zu dem als 100 festgelegten Ausgangswert des Serums Wie die Tabelle zeigt, bestätigen diese Versuche sehr deutlich die schon mit $Jod^{131}$-markiertem Humanalbumin gefundenen Ergebnisse, so daß wir

TABELLE 8 *Vergleich der Aktivitätskonzentration in Serum, Liquor und Perilymphe (Fraktion I und II) nach suboccipitaler Applikation von $Jod^{131}$-markierten Humanalbuminen und kanincheneigenen Albuminen (Mittelwerte).*

| Zahl d Fälle | Vers Dauer | Aktivität (= Impulszahl/25 µl) in % des Ausgangswertes | | | |
|---|---|---|---|---|---|
| | | Serum | Liquor | Perilymphe Fraktion I | Perilymphe Fraktion II |
| 3 | 10 Min | — | 100 | 44,7 | 50,0 |
| Kaninchen Albumin | 10 Min | — | 100 | 17,3 | — |
| 2 | 72 Std | 100 | 7,0 | 20,5 | 10,9 |
| 2 | 92 Std | 100 | 3,9 | 55,0 | 17,4 |

Tabelle 6. *Vergleich der Aktivitätskonzentration in Serum, Liquor und Perilymphe (Fraktion I und II) nach intravenöser Applikation von Jod$^{131}$-markiertem Human-Albumin (Mittelwerte).*

| Zahl d. Fälle | Vers. Dauer | Aktivität (= Impulszahl/25 µl) in % des Ausgangswertes: Serum | Liquor | Perilymphe Fraktion I | Perilymphe Fraktion II |
|---|---|---|---|---|---|
| 5 | 10 Min | 100 | 0,11 | 0,13 | 0,15 |
| 6 | 60 Min | 72 | 0,13 | 0,22 | 0,23 |
| 7 | 220 Min | 68 | 0,15 | 0,20 | 0,29 |
| 8 | 48 Std | 20 | 0,16 | 0,31 | 0,13 |

überein und die methodische Fehlerbreite von $\pm 3\%$ wurde nicht überschritten.

Die *Messungen* der Aktivitäten führten wir mit dem Strahlungsmeßgerät FH 90 — Szintillationszähler mit Bohrlochkristall FH 451 durch.

Folgende Versuche wurden vorgenommen:

a) 22 Kaninchen erhielten je 100 µC Humanalbumin und 3 Kaninchen kanincheneigenes jodmarkiertes Albumin in die Ohrvene injiziert. Die Versuchsdauer betrug 10 Minuten bis 48 Stunden. Nach 10 Minuten wurde aus der Vene des anderen Ohres ein Tropfen Blut und Blut für die Serumgewinnung als Bezugswert abgenommen.

Zur Perilymphgewinnung präparierten wir am narkotisierten Tier das Innenohr nach der angegebenen Methode und entnahmen die beiden Fraktionen, die in je 1 Meßschälchen gegeben wurden. Anschließend punktierten wir die Zisterna magna zur Liquorgewinnung. Schließlich töteten wir das Tier und entnahmen aus dem Herzen Blut zur Direktmessung und Blut zur Serumgewinnung. Gleiche Mengen von den einzelnen Körperflüssigkeiten wurden jeweils in 1 Meßschälchen abpipettiert.

In der Tabelle 6 sind die Ergebnisse dieser Versuchsanordnung aufgezeichnet.

Die Aktivität ist diesmal ausgedrückt in Quotienten aus Impulszahlen pro 25 µl untersuchte Körperflüssigkeit, bezogen auf den 10 Minuten Ausgangswert des Serums.

Eine Aktivität war frühestens nach 10 Minuten in der Perilymphe nachweisbar. Die Aktivität des Liquors lag zu diesem Zeitpunkt noch wesentlich unter der Aktivität der Fraktion I der Perilymphe, während die Aktivitätskonzentration in der Fraktion II nur etwas höher war als die Konzentration in der gleichen Menge Liquor. Nach 60 Minuten verringerte sich der Abstand zwischen der Konzentration im Liquor und in der Perilymphe. Die Aktivität in der Perilymphe war nur noch doppelt so groß wie im Li-q[illegible], jedoch war die Konzentration in der nachgeflossenen Perilymphe ([illegible]tion II) höher als in der Fraktion I. Nach 2 Stunden war das Ergebnis

Es zeigten sich die in Tabelle 9 angegebenen Ergebnisse

Wie aus der Tabelle zu ersehen ist, fand sich nach kurzer Zeit in der Perilymphe des Ohres mit verschlossenem Aquaeductus cochleae eine um 100 fach geringere Aktivität als in der Perilymphe des anderen Ohres, wenn die aktive Substanz suboccipital appliziert wurde

Dagegen war die Aktivitätskonzentration nach intravenöser Applikation in der Perilymphe des Ohres mit verschlossenem Aquaeductus cochleae nur um ein Drittel geringer als in der Perilymphe des Ohres mit offenem Aquaeductus cochleae Auf dieses Ergebnis wird bei der Besprechung der Befunde noch besonders eingegangen

d) In 3 Fällen applizierten wir zur Prüfung der Durchlässigkeit der Reißnerschen Membran das markierte Albumin in den Perilymphraum und gewannen anschließend nach der schon oben angegebenen Methode die Endolymphe

Es zeigt sich, daß 10 Minuten nach Applikation der Endolymphe keine Aktivität nachzuweisen war Auch bis zu einem Zeitraum von 1 Stunde war keine Aktivität in der Endolymphe zu finden Ebenso war auch keine Aktivität in der Perilymphe des anderen Ohres feststellbar

3 *Autoradiographische Untersuchungen*[1]

a) *Tierversuche* 2 Kaninchen mit einem Gewicht von 2650 g und 2810 g, die 1 Tag nüchtern waren, erhielten je 70 mC tritiummarkiertes Phenylalanin in die Ohrlöffelvene injiziert Die spezifische Aktivität betrug 8000 mC/mMol Bei 2 Kaninchen mit einem Durchschnittsgewicht von 2720 g wurden je 90 mC tritiummarkiertes Lysin mit einer spezifischen Aktivität von 3000 mC/mMol intravenös verabreicht Mittels einer Überdosis Evipan töteten wir die Kaninchen je einer Versuchsreihe nach 30 und 60 Minuten Anschließend wurden die Innenohren beiderseits entnommen und die Präparate in 10%igem Formalin fixiert, dem noch inaktives Phenylalanin, bzw Lysin zugesetzt war Die Präparate wurden in 5%iger Salpetersäure entkalkt

b) *Technik der Autoradiographie* Nach der üblichen Einbettung in Paraffin wurden 5–6 $\mu$ dicke Serienschnitte angefertigt, die teils zur Autoradiographie, teils zur Haematoxilin Eosinfärbung verwendet wurden

Die zur Autoradiographie bestimmten Schnitte wurden auf fettfreien Objektträgern mit Eiweißglyzerin aufgeklebt Nach Entparaffinierung überzogen wir diese mit einer 2–3 $\mu$ dicken Gelatineschicht (0,5%ige Gelatinelösung mit Zusatz von 0,05%igem Chromalaun)

Bei fast völliger Dunkelheit wurden die Präparate nach dem von PELC (1947), BOYD (1948) und MAURER (1959) angegebenen Stripping Film-Verfahren mit einem Kodak-Autoradiographic Film AR 10 (Emulsionsdicke 5 $\mu$) bedeckt und an der Luft getrocknet Die Präparate wurden 24 Stunden bis 3 Monate exponiert Nach Abschluß der Exposition entwickelten wir die

[1] Herrn Dr Dr Hempel Institut für med Isotopenforschung in Köln (Leiter Prof W Maurer) danken wir für die Herstellung der tritiummarkierten Aminosäuren

Präparate unter konstanten Bedingungen und danach wurde ein Teil durch die Filmschicht hindurch mit Haemalaun gefärbt. Ein anderer Teil der Autoradiogramme blieb ungefärbt. Das Eindecken der Präparate erfolgte mit Glyzeringelatine.

c) *Ergebnisse der autoradiographischen Untersuchungen.* Die quantitative Auswertung der Autoradiogramme (ARG) geschah durch die Ermittlung der Silberkorndichte über den einzelnen Gewebs- und Zellarten.

Für die Silberkornzählung wurden schwachbelichtete ARG genommen, wobei wir die Silberkörner pro $\mu^2$ Gewebsfläche in einem Ocularnetzmikrometer auszählten. Das Einzelquadrat entsprach bei Oelimmersion einer Fläche von 16 $\mu^2$.

Durch die Verwendung von $H^3$-markierten Aminosäuren konnte ein gutes Auflösungsvermögen erzielt werden, das nach MAURER (1959) unter 1 $\mu$ liegt, so daß die Silberkörner (SK) den in den 5 $\mu$ dicken Schnitten liegenden Zellen mittels starker Vergrößerung ohne Schwierigkeiten zugeordnet werden konnten. Bei SK-Zahlen unter 20 und weniger pro Zellquerschnitt genügte ein einmaliges Auszählen, bei größeren SK-Zahlen wurde zur Kontrolle nochmals ausgezählt.

*Die in den Abbildungen und Tabellen für $H^3$-Lysin und $H^3$-Phenyl-Alanin* wiedergegebenen Ergebnisse wurden für die jeweilige Substanz an ein und demselben ARG gewonnen. Die Ergebnisse sind deshalb miteinander vergleichbar. Bei unterschiedlichen Belichtungszeiten war die SK-Zahl der Belichtungszeit proportional. MAURER (1959) fand, daß die absoluten Werte der SK-Zahlen unter anderem abhängen von der jeweils applizierten Aminosäureaktivität und der Belichtungszeit der ARG. In unseren Versuchen sind deshalb nicht die absoluten SK-Zahlen, sondern lediglich die Zahlenverhältnisse *derselben bei den einzelnen Zellarten* vergleichbar.

Die autoradiographisch ermittelte SK-Dichte ist nach MAURER (1959) ein lineares Maß für die lokalspezifische Umsatzrate an der betreffenden Stelle des Schnittes. Die Schwärzungsverteilung im ARG gibt also die Größe der lokalen Aminosäureinkorporation wieder und durch Auszählung der SK pro $\mu^2$ Gewebsfläche ist ein quantitativer Vergleich des Aminosäureeinbaues in den einzelnen Geweben und Zellen möglich.

In der Tabelle 10 sind die Auswertungsergebnisse bei der Schnecke nach intravenöser *Injektion von $H^3$-Phenyl-Alanin* wiedergegeben. Die Versuchsdauer betrug 30 und 60 Minuten.

Die stärkste SK-Dichte weist die Ganglienzelle des Ganglion spirale cochleae auf, während die Schwärzungsverteilung in allen anderen Geweben in der Reihenfolge Tractus spiralis arteriosus et venosus, Stria vascularis, Ligamentum spirale, Reißnersche Membran, Corti'sches Organ und Membrana tectoria abnimmt.

Die Abbildung 6 zeigt eine Lupenvergrößerung eines überbelichteten Autoradiogramms (ARG) der Schnecke. Der unter der Fotoemulsion liegende Schnitt wurde nicht gefärbt. Man erkennt sehr deutlich die unter-

[1] Schwärzungsverteilung in den einzelnen Geweben der Cochlea

TABELLE 10 *Mittlere Silberkorndichte über den Geweben und Zellen der Cochlea und relative Einbaurate nach intravenöser Applikation von H³-Phenyl-Alanin beim Kaninchen nach einer Versuchsdauer von 30 und 60 Minuten*

| Art des Gewebes oder der Zellen | Versuchsdauer 30 Minuten | | Versuchsdauer 60 Minuten | |
|---|---|---|---|---|
| | Silberkörner pro $\mu^2$ | Relative Einbaurate (Ganglienzellen = 100) | Silberkörner pro $\mu^2$ | Relative Einbaurate (Ganglienzellen = 100) |
| Ganglienzellen des Ggl spirale | 2,38 | 100 | 2,38 | 100 |
| Bindegewebszellen des Modiolus | 1,89 | 79 | 1,89 | 79 |
| Tractus arteriosus et venosus | 2,28 | 96 | 2,28 | 96 |
| Stria vascularis | 1,76 | 67 | 1,76 | 67 |
| Cortisches Organ (Haar und Stützzellen) | 0,91 | 38 | 0,98 | 42 |
| Reißnersche Membran | 0,76 | 32 | 0,90 | 36 |
| Membrana tectoria | 0,41 | 17 | 0,45 | 19 |
| Tympanale Beleg Zellen | 0,74 | 31 | 0,91 | 37 |
| Bindegewebszellen des Ligamentum spirale unterhalb und oberhalb der Reißnerschen Membran | 1,38 | 58 | 1,38 | 58 |
| Bindegewebszellen des Limbus spirale | 1,24 | 52 | 1,24 | 52 |

TABELLE 11 *Mittlere Silberkorndichte über den Geweben und Zellen der Cochlea und relative Einbaurate nach intravenöser Applikation von H³-Phenyl-Alanin beim Kaninchen nach einer Versuchsdauer von 30 und 60 Minuten*

| Art des Gewebes oder der Zellen | Versuchsdauer 30 Minuten | | Versuchsdauer 60 Minuten | |
|---|---|---|---|---|
| | Silberkörner pro $\mu^2$ | Relative Einbaurate (Ganglienzellen = 100) | Silberkörner pro $\mu^2$ | Relative Einbaurate (Ganglienzellen = 100) |
| Ganglienzellen des Ggl vestibuli | 2,86 | 100 | 2,86 | 100 |
| Bindegewebszellen der Labyrinthwandungen | 1,66 | 58 | 1,70 | 59 |
| Neuroepithel der Macula (Haar und Stützzellenschicht) | 1,40 | 49 | 1,59 | 56 |
| Bogengangsepithel | 1,21 | 43 | 1,36 | 48 |
| Otolithenmembran | 0,51 | 18 | 0,69 | 23 |
| Neuroepithel der Crista ampullaris (Haar und Stützzellenschicht) | 1,37 | 48 | 1,63 | 56 |
| Übergangszone und Planum semilunatum | 1,74 | 61 | 1,77 | 61 |
| Cupula | 0,49 | 17 | 0,61 | 22 |

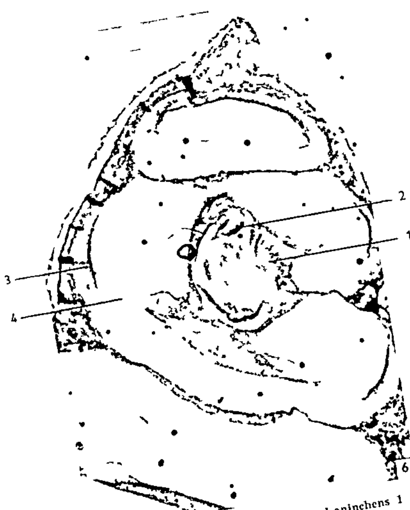



Abb 6 Ungefarbtes Autoradiogramm der Cochlea des Kaninchens 1 Stunde nach Gabe von H³ Phenyl Alanin (Stripping Film 30 fach) Die starkste Schwarzung ist uber den Ganglienzellen des Ggl spirale cochleae (1) dann folgt der Tractus arteriosus et venosus (2) die Stria vascularis (3) und das Corti sche Organ (4)

Wie unsere Tabelle zeigt wiesen einige Gewebe der Cochlea schon nach 30 Minuten ihre endgultige Aminosaure Inkorporation auf, wahrend bei einigen anderen Geweben erst nach 60 Minuten der Hochstwert der Silber korndichte erreicht ist Diese Befunde decken sich hierin weitgehend mit denjenigen von MEYER ZUM GOTTESBERGE (1960) PLESTER (1960) und KOBURG und PLESTER (1962) am Meerschweinchen ermittelten

Abbildung 7 gibt ein ARG des Ductus cochlearis wieder Die Stria vascularis weist mit einer Silberkorndichte von 1 76 Sk/$\mu^2$ einen ihrer Funktion entsprechenden starken Eiweißstoffwechsel auf

Im Corti schen Organ besteht kein Unterschied in der Silberkorndichte [illegible] Haarzellen und Stutzzellen Die Basilarmembran ist praktisch [illegible] Silberkornern wahrend die Membrana tectoria nur einen geringen




Abb. 7. Autoradiogramm des Ductus cochlearis beim Kaninchen 1 Stunde nach Applikation von $H^3$-Phenyl-Alanin (Stripping-Film, 90fach, gefärbt). Stärkste Silberkorndichte in der Stria vascularis (X)




Abb. 8. Ungefärbtes Autoradiogramm des Cortischen Organs (Stripping-Film, 300fach). Die Basilarmembran (1) und die Membrana tectoria (2) zeigen nur eine ganz geringe Schwärzungsverteilung. Starke Schwärzung der *tympanalen Belegschicht* (3)

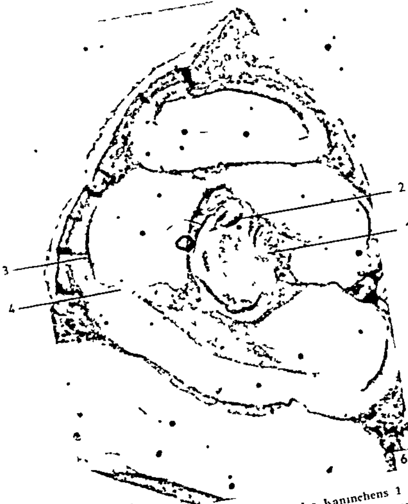



ABB 6 Ungefarbtes Autoradiogramm der Cochlea des Kaninchens 1 Stunde nach Gabe von H³ Phenyl Alanin (Stripping Film 30 fach) Die starkste Schwarzung ist uber den Ganglienzellen des Ggl spirale cochleae (1), dann folgt der Tractus arteriosus et venosus (2) die Stria vascularis (3) und das Corti sche Organ (4)

Wie unsere Tabelle zeigt, wiesen einige Gewebe der Cochlea schon nach 30 Minuten ihre endgultige Aminosäure-Inkorporation auf, wahrend bei einigen anderen Geweben erst nach 60 Minuten der Hochstwert der Silberkorndichte erreicht ist Diese Befunde decken sich hierin weitgehend mit denjenigen von MEYER ZUM GOTTESBERGE (1960), PLESTER (1960) und KOBURG und PLESTER (1962) am Meerschweinchen ermittelten

Abbildung 7 gibt ein ARG des Ductus cochlearis wieder Die Stria vascularis weist mit einer Silberkorndichte von 1,76 Sk/$\mu^2$ einen ihrer Funktion entsprechenden starken Eiweißstoffwechsel auf

Im Corti schen Organ besteht kein Unterschied in der Silberkorndichte [illegible] Haarzellen und Stutzellen Die Basilarmembran ist praktisch [illegible] ern wahrend die Membrana tectoria nur einen geringen [illegible] n Si [illegible]




Abb 10 Ungefärbtes Autoradiogramm der Crista ampullaris mit Cupula 1 Stunde nach Gaben von H³ Phenyl-Alanin (Stripping Film, 90 fach) Die Cupula zeigt nur wenig Silberkörner

SK/$\mu^2$) Fast unbedeutend war der Eiweißstoffwechsel in der Otolithenmembran und der Cupula (0,31 SK/$\mu^2$ bzw. 0,49 SK/$\mu^2$) Die Cupula war nur in wenigen histologischen Schnitten erhalten, es ist bekannt, daß ihre Darstellung technisch große Schwierigkeiten bereitet

Abb 9 gibt ein ungefärbtes ARG der Crista ampullaris und der gleichzeitig mitgetroffenen Macula wieder Aus der Abbildung ist ersichtlich, daß die größte Schwärzung im Bereich der Übergangszone und im Ampullenepithel festzustellen war Diese Schwärzung geht aber zum Teil auf das hier eingelagerte Pigment zurück Die SK-Dichte nimmt in der Reihenfolge Neuroepithel, Bogengangepithel, Otolithenmembran und Cupula ab

Auffallend ist eine stärkere SK-Dichte im Bereich der Macula zwischen Neuroepithel und Otolithenmembran

Vergleicht man anhand der Tabelle Nr 11 die Werte des 30 Minutenver-

suches mit denjenigen des Versuches nach 60 Minuten, so ergibt sich, daß die Sk-Dichte in den Ganglienzellen, dem Randepithel des Planum semilunatum und im Gefäßbindegewebe der Labyrinthwandungen nach 60 Minuten nicht zugenommen hat. Dagegen ist ein Ansteigen der Sk-Dichte bei dem Neureepithel sowie beim Bogengangsepithel und der Otolithenmembran und Cupula festzustellen.

Abb. 10 zeigt ein ungefärbtes ARG der Crista ampullaris mit Cupula. Man erkennt sehr deutlich, wie gering die Sk-Dichte im Bereich der Cupula gegenüber dem Neuroepithel ist.

Bei der zweiten Versuchsserie, bei der wir H³-Lysin injizierten, zeigte sich bei der Auswertung der ARG, daß die relative Einbaurate der Aminosäure sowohl im Bereich der Cochleae als auch im Vorhofbogengangapparat dieselben Werte erreichte, wie nach der Applikation von H 3-Phenyl-Alanin. Wir verzichteten deshalb auf eine Wiedergabe der Tabellen. Der Befund, daß trotz Anwendung verschiedener Aminosäuren im wesentlichen dieselbe Schwärzungsverteilung festzustellen war, deckte sich mit Ergebnissen, wie sie MAURER u. Mitarb. (1959) auch bei anderen Organen aufweisen konnten.

# III. BESPRECHUNG DER ERGEBNISSE

Bei der chemischen Analyse der Perilymphe hat sich ergeben, daß hinsichtlich des Elektrolytgehaltes nur ein geringer Unterschied in der Zusammensetzung gegenüber dem Liquor cerebrospinalis besteht. Der Natriumgehalt ist etwas geringer als im Liquor, dagegen ist der Kaliumgehalt ähnlich dem des Liquors, während das Kalzium in allen Körperflüssigkeiten in ziemlich gleicher Konzentration vorhanden ist. Unsere Befunde decken sich hierin mit den Ergebnissen, die von SMITH, LOWRY und WU (1954) und von CITRON und EXLEY (1957) in der Perilymphe von Meerschweinchen sowie von RAUCH (1958) in menschlicher Perilymphe (aus Leichenfelsenbeinen gewonnen) gefunden wurden.

ANTONINI, CASORATI und CRISO (1957) untersuchten als erste die Konzentration der Aminosäure in der Perilymphe. CHEVANCE, GALLI und JEANMAIRE (1960) führten erstmals Immunoelektrophoresen durch und gewannen verschiedene Proteinfraktionen. Sie verglichen ihre Ergebnisse an der Perilymphe mit dem Verteilungsmuster des Liquors, und es ergaben sich hierin Unterschiede.

In 22 eigenen Versuchen haben wir erstmals 2 Fraktionen der Perilymphe beim Kaninchen papierchromatographisch auf ihren Gehalt an Aminosäuren untersucht. Wie wir schon bei der Methode zur operativen Perilymphgewinnung hervorheben konnten, bildet sich nach Gewinnung der Perilymphe im Perilymphraum (Fraktion I) nach einigen Minuten wieder Perilymphe (Fraktion II) nach. Es war für uns von Interesse, ob diese Flüssigkeit (Fraktion II) dieselbe Zusammensetzung hat wie die Fraktion I. Wir konnten feststellen, daß die Fraktionen I und II hinsichtlich der freien Aminosäuren rein qualitativ in der Zusammensetzung gegenüber dem Liquor fast gleich ist. Hinsichtlich des absoluten Gehaltes zeigt sich, *daß die Fraktion II nur die Hälfte an freien Aminosäuren aufweist* wie die Fraktion I, Liquor und Blutserum. Darüber hinaus war festzustellen, daß für den Vergleich von Perilymphe Fraktion I und II gegenüber Liquor und Serum für die einzelnen isolierten Aminosäuren in unterschiedlicher Weise für Serum oder Liquor charakteristische Werte in der Perilymphe auftreten.

Unterschiede hinsichtlich der Zusammensetzung finden sich auch beim Vergleich des Eiweißgehaltes von Liquor und Perilymphe. In letzterer ist der Eiweißgehalt etwa 4- bis 5mal so hoch als im Liquor.

Es gelang uns, die Perilymphe auch elektrophoretisch *aufzuspalten*. Gegenüber den Ergebnissen am Serum und Liquor zeigten sich hier Unterschiede. Es fiel vor allem der höhere Albumingehalt auf.

Aus den bisherigen Befunden ergibt sich, daß sich die Perilymphe in ihrer chemischen Zusammensetzung von der des Liquors unterscheidet. Unsere Untersuchungen bestätigen damit die Befunde, die andere Autoren beim Meerschweinchen, bei der Katze und beim Menschen gefunden haben. Die papierchromatische Untersuchung zeigt darüber hinaus, daß die beiden Fraktionen I und II der Perilymphe nicht der gleichen Bildungsstätte entspringen können.

Zur Klärung der Frage, ob es sich bei der Perilymphe um eine selbständige Flüssigkeit handelt, haben wir uns dem Studium der Bildungsstätte und der Herkunft dieser Labyrinthflüssigkeit gewidmet. Bei Versuchen, die zu diesem Problem von anderen Autoren früher durchgeführt worden sind, stand vor allem die Frage der Durchgängigkeit des Aquaeductus cochleae im Mittelpunkt des Interesses.

WITTMAACK (1936) sah in dem Aquaeductus cochleae noch eine offene Verbindung zum Schädelinneren und vertrat die Ansicht, daß die Perilymphe durch diesen offenen Kanal aus dem Perilymphraum in den Liquorraum abfließt. Er berichtete auch über echte Konkremente im Aquaeductus cochleae (Aq. c.), die zufällig bei der Untersuchung des Felsenbeines eines Patienten gefunden wurden, der zu Lebzeiten an Menière-schen Anfällen gelitten hatte. WITTMAACK nahm deshalb an, daß eine Verlegung des Aq. c. zu Druckdifferenzen innerhalb des Labyrinthes führt.

Klinische Beobachtungen bei der Stapeschirurgie (MÜNDNICH 1959, UNGERECHT 1960) führten auch zu der Annahme, daß zumindest vereinzelt eine offene Verbindung zwischen dem Liquorraum und dem Perilymphraum beim Menschen vorhanden sein müsse. So konnten diese Autoren feststellen, daß sich bei Entfernung der Stapesfußplatte in seltenen Fällen plötzlich unter Druck bis zu mehreren 100 ccm Labyrinthliquor entleerte. Chemische Untersuchungen ergaben (SCHREINER 1962, unveröffentlicht), daß es sich hierbei um Liquor cerebrospinalis handelt. Ob dieser Liquor durch einen offenen Aq. c. oder durch perivasale oder perineurale Kanäle etwa entlang des Facialiskanales im inneren Gehörgang (RAUCH 1962) in das Innenohr gelangt, bleibt solange ungeklärt, bis es nicht gelingt, die eine oder andere Annahme durch Nachweis einer Mißbildung histologisch zu sichern.

Bei experimentellen Versuchen über die Durchgängigkeit des Aq. c. am Tier injizierten die Autoren die verschiedensten Testflüssigkeiten, wie Gelatine und Tinte (WEBER-LIEL 1879, JAMPOLSKI 1925), Tusche (WITTMAACK 1917, ARNVIG 1947), Kaliumferrocyanid, das sich mit Eisenammoniumnitrat blau färbt (GUILD 1927, MEURMANN 1931, ALTMANN und WALTNER 1947, SVANE-KNUDSEN 1958), teils in den Subarachnoidalraum, teils subcutan oder direkt in die Blutbahn. Auch Methylenblau (OHMA 1939) und besonders Fluoreszin (VENTURA GREGORINI 1955, GISSELSSON 1949, [illegible] und PORETTI 1950, KLEY 1951 und JAKO und Mitarb. 1959) wurde viel [illegible]. Viele Autoren waren sich hierbei bewußt, daß sie durch die [illegible]er Substanzen die physiologischen Verhältnisse im Innenohr [illegible] untersuchenden Flüssigkeiten stören, es ist deshalb nicht

verwunderlich, wenn die Ergebnisse von Autor zu Autor sehr variierten. Weitere Aufklärungen brachten über den Grund dieser unterschiedlichen Untersuchungsergebnisse schließlich histologische Untersuchungen von WALTNER und ALTMANN (1947). Sie fanden beim Kaninchen, bei menschlichen Föten und bei einigen Felsenbeinen vom erwachsenen Menschen eine kontinuierliche Membran, die die Öffnung des Aq. c. von der Scala tympani abtrennt. Der Aq. c. hat nach Messungen dieser Autoren beim Kaninchen eine durchschnittliche Länge von 2,5–3 mm und eine durchschnittliche Weite von 0,3–0,5 mm. Gegen die innere und äußere Öffnung wird der Kanal etwas weiter. Im Inneren ist der Kanal mit einem Netzwerk retikulären Bindewebes ausgefüllt. Ein Vergleich des Aq. c. beim Kaninchen mit dem des Menschen zeigt, daß beim Kaninchen der Kanal sehr viel weiter als beim Menschen ist. Viel wichtiger aber ist noch das Verhältnis zwischen Länge und Weite. Beim Kaninchen ist es etwa 7 : 1 und beim Menschen 100 : 1. ALTMANN und WALTNER (1947/48) kamen zu dem Schluß, daß die von ihnen gefundene Trennmembran eine komplette Schranke zwischen dem Liquorraum und dem Perilymphraum darstellt.

LEMPERT und Mitarb. (1952) vermuten, daß nicht die WALTNER'sche Membran, sondern das enge Lumen des Aq. c. und die Schwellung des bindegewebigen Netzwerkes einen Flüssigkeitsaustausch verhindere. KLEY (1951) konnte an Felsenbeinen menschlicher Leichen nur mit hohem, plötzlich einsetzenden Druck (55 mm Hg) einen Flussigkeitsdurchtritt durch den Aq. c. erzwingen. Aus diesem Grunde hätten entzündliche Labyrintherkrankungen auch nicht ohne weiteres eine Meningitis zur Folge und umgekehrt beteilige eine Meningitis praktisch nicht das Labyrinth. Derselbe Autor kommt aber auf Grund seiner Versuche beim Tier zu dem Ergebnis, daß der Aq. c. durchgängig ist und er hält auch einen gewissen Zufluß von Liquor entlang der perineuralen Lymphscheiden uber dem inneren Gehörgang und den Modiolus fur möglich. Auch eine Bildung von Perilymphe im Perilymphraum selbst oder aus der Endolymphe komme infrage.

HOMMERICH (1961) konnte feststellen, daß eine Druckubertragung auf das Labyrinth nicht durch eine Erhöhung des Liquordruckes, der sich auf den Perilymphraum fortsetzen konnte, geschehe, sondern nur dann eintrete, wenn der „trockene Hirndruck" auf die lateralen Bruckenzisternen zur Auswirkung kommt.

Zieht man in Betracht, daß der Aq. c. von Tier zu Tier der gleichen Gattung und noch mehr von Tier zu Tier einer anderen Gattung große Unterschiede aufweist, und berucksichtigt man, daß durch die meisten Versuche die physiologischen Verhältnisse mehr oder weniger gestört wurden, dann ist verständlich, daß als Voraussetzung jeder weiteren Untersuchung, die der Klarung des Problems über die Herkunft der Labyrinthflüssigkeiten dienen soll, diese Fehlerquellen nach Möglichkeit ausgeschaltet werden mussen. Aus diesen Gründen ist den Versuchen mit radioaktiven Stoffen eine besondere Bedeutung beizumessen. Die Radioisotope verhalten sich biologisch und chemisch wie die stabilen Nuklide desselben Elementes,

sind nicht körperfremd, stören nicht das osmotische Gleichgewicht und sind vor allem in kleinsten Mengen physikalisch nachweisbar

GRAF und PORETTI (1950), sowie PORTMANN (1954) injizierten Phosphor-32 subkutan oder intraperitoneal und fanden, daß er später in der Perilymphe auftrat als im Kammerwasser oder im Liquor GRAF und PORETTI (1950) führten diese geringe Radioaktivität auf die kleinere Austauschfläche zwischen Blut und Perilymphe einerseits und Blut und Liquor andererseits zurück Bei Versuchen mit Natrium 24 erschien die aktive Substanz jedoch fast gleichzeitig in der Perilymphe und im Liquor Die Autoren schlossen daraus, daß die Perilymphe ein Blutdialysat sein könnte

RAUCH (1960) führte Untersuchungen mit radioaktiver $^{24}$NaCl-Lösung beim Meerschweinchen durch und fand, daß 18 Stunden nach Applikation die Radioaktivität in den parenchymatösen Organen geringer war als in der Peri- und Endolymphe

Bei unseren Traceruntersuchungen mit radioaktiven Stoffen wollten wir anhand des Vergleiches der Konzentration in der Perilymphe mit derjenigen im Blut und Liquor feststellen, ob sich Rückschlusse uber den Weg des Antransportes der radioaktiven Substanzen zu den jeweiligen Körperflussigkeiten gewinnen lassen Insbesondere sollte dabei gepruft werden, inwieweit sich die Konzentrationen der Radioaktivität in Abhängigkeit von der Zeit nach der Injektion ändern

Im Falle der intravenösen Injektion von radioaktivem Phosphor laßt sich schon 5 Minuten später eine Radioaktivität in der Perilymphe nachweisen Die Aktivität im Blut ist sehr hoch und fallt in der Folgezeit kontinuierlich ab Es tritt allmählich ein Ausgleich der Aktivitäten zum Liquorraum und ein entsprechender Ausgleich mit der Perilymphe ein. Nach langen Zeiträumen ist die Aktivität in der Perilymphe fixiert Entsprechende Ergebnisse finden sich auch bei Injektionen in den Liquorraum, wobei ein rascher Aktivitätsausgleich zu Perilymphe erfolgt Der größte Teil der Liquoraktivität geht uber den Kreislauf und das Ausscheidungssystem verloren Nach 24 Stunden sind die Aktivitaten von Liquor und Perilymphe ausgeglichen

Bei subocciptaler Applikation ist der Aktivitätseintritt nur auf direktem Wege und nicht uber das Kapillarsystem möglich Ein Vergleich bei der subocciptalen und intravenösen Applikation von Phosphor-32 ergibt fast die gleichen Konzentrationsverhaltnisse Es ist anzunehmen, daß niedermolekulare Substanzen die Perilymphe sowohl über den Blutweg als auch uber den Liquorweg erreichen können. Fur die Annahme, daß Phosphor-32 direkt vom Blut aus in den Perilymphraum eingeschleußt wird spricht der Befund daß 5 Minuten nach intravenöser Applikation nicht schon eine so hohe Aktivität von Phosphor in der Perilymphe nachzuweisen ist Die Diffusion von aktivem Phosphor aus dem Blut uber den Liquorraum in den Perilymphraum wurde länger als 5 Minuten dauern

Es ist hervorzuheben, daß selbst bei völlig gleicher Dosierung von radioaktivem Phosphor sich in Abhängigkeit von physiologischen Faktoren von

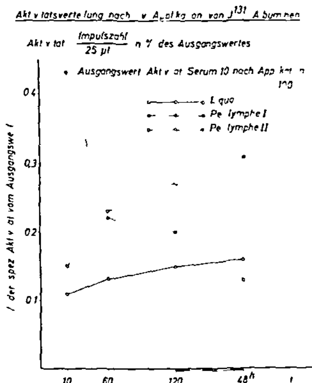



ABB 11 Die graphische Darstellung der Aktivitätskonzentrationen in Liquor und Perilymphe als Funktion der Zeit nach intravenöser Applikation von Jod[131] markierten Albuminen (Mittelwerte)

Versuchstier zu Versuchstier individuelle Schwankungen hinsichtlich der Ausscheidung der peripheren Durchblutung und damit des Abstromens in die Organe sowie in der Permeabilität der Blutliquorschranke ergaben Dazu kommt daß das gesamte Blutvolumen eine geringe Austauschfläche mit dem Liquor besitzt und daß die Perilymphe entsprechend eine noch kleinere ionendurchgängige Membran zum Liquor aufweist Es ist zu berücksichtigen daß Phosphor 32 zum Teil organisch gebunden werden kann und damit der Diffusion entzogen wird Es ist möglich daß die geringere *Aktivitätskonzentration in der Perilymphe nach 5 und 15 Minuten gegenüber* dem Liquor auf diese Faktoren zurückzuführen ist

Zur weiteren Klärung unserer Fragestellung haben wir Versuche mit einem extrazellulären Stoff nämlich dem Jod 131 radioaktiv markiertem Albumin durchgeführt Das verwendete Albumin hat eine Molekulargröße von ca 60 000

Wir applizierten diesen Indikator intravenös suboccipital und in den Perilymphraum Wie das obige Diagramm (Abb 11) zeigt ist 10 Minuten nach intravenöser Injektion erstmals eine Radioaktivität in der Perilymphe nachweisbar Die Aktivitätskonzentration ist in der Fraktion I etwa 4 mal so hoch wie im Liquor Die nachgeflossene Perilymphe (Fraktion II)

entspricht in ihrer Aktivitätskonzentration etwa den Werten des Liquors Nach 1 Stunde ist die Konzentration in der Perilymphe der Fraktion I nicht so hoch wie im Liquor Die Konzentration im Liquor steigt auch absolut an, während sie in der Perilymphe auf die Hälfte fällt Die Fraktion II ist sogar etwas hoher als die Fraktion I Die Aktivitätskonzentration im Blut nimmt um ein Drittel ab Nach ca 2 Stunden sind die Werte ähnlich dem des 1-Stundenversuches, nur ist die Verschiebung zugunsten der Aktivitätskonzentration noch deutlicher Nach längeren Zeiträumen, bis zu 48 Stunden, ist die Aktivität in der Perilymphe am höchsten, während sie im Liquor und in der Fraktion II der Perilymphe fast gleich ist

Aus diesen Ergebnissen ist zu schliessen, daß sehr enge Beziehungen zwischen dem Blutkapillarsystem und der Perilymphe bestehen Die Befunde zeigen, daß radioaktiv markiertes Albumin direkt aus dem Kapillarsystem in den Perilymphraum eintreten muß Andererseits aber bestehen auch enge Beziehungen zwischen dem Liquorraum und der Perilymphe Die Aktivitätskonzentrationen in der Fraktion II der Perilymphe weisen darauf hin, daß bei längerer Versuchsanordnung auch noch ein Nachschub aus dem Liquorraum in den Perilymphraum erfolgt

Wird das radioaktiv markierte Humanalbumin subocciptal appliziert so findet man nach 10 Minuten schon fast die Hälfte der Radioaktivität in der Perilymphe (Abb 12)

Wie das Diagramm zeigt, kommt in der Aktivitätskonzentration der Fraktion II der Perilymphe deutlich zum Ausdruck, daß es sich bei letzterer nur um nachgeflossenen Liquor handeln kann Dafur spricht auch, daß bei Versuchen mit langerer Dauer die Aktivität im Liquor stark abfällt, während sich die Aktivität im Perilymphraum verändert

Aufgrund der anatomischen Verhältnisse kommt der Aquaeductus cochleae als direkter Verbindungskanal zwischen Perilymph- und Subarachnoidalraum infrage Wir haben diesen Kanal auf der einen Seite plombiert und dann die radioaktive Substanz subocciptal appliziert Anschließend wurde die Aktivität in der nachgelaufenen Perilymphe der operierten Seite und der Perilymphe der nicht plombierten anderen Seite gemessen Beim Vergleich (Tab 9) findet sich eine bis zu 100-fach höhere Aktivität in der Perilymphe des Ohres mit nicht plombiertem Aq c als auf der plombierten Seite Auch diese Versuche sprechen fur eine direkte Einwanderung von Liquor in den Perilymphraum uber den Aq c

Ein Nachströmen des Liquors muß aber noch über andere Wege als über diesen Kanal erfolgen Dies beweisen die Versuche, daß auch bei verschlossenem Aq c noch Aktivität in der Perilymphe nachzuweisen war Wir nehmen an daß dieses Einströmen von Liquor in den Perilymphraum uber die perineuralen Lymphscheiden möglich ist, wofür auch die Untersuchungen von KLEY (1951) mit Eisenfarbstoffen sprechen

W[illegible] wir an anderer Stelle zeigen konnten (SCHREINER 1963), besteht je-[illegible] [illegible]rschied ob man die Perilymphe im geöffneten Innenohr, also [illegible] nicht eröffneten Innenohr, also nach der Gefriermethode,

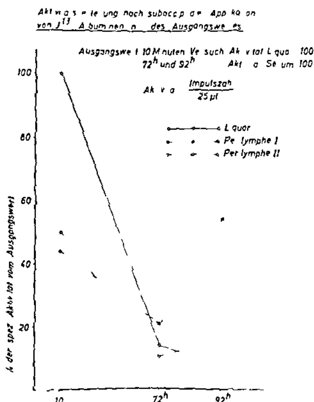



Abb 12 Diagramm der Aktivitatskonzentrationen in Liquor und Perilymphe als Funktion der Zeit nach subocipitaler Applikation von J 1[31] markierten Albuminen (Mittelwerte)

gewinnt Die Befunde geben Hinweise daß der Aq e offenbar nur bei unphysiologischen Verhältnissen, d h bei veränderten Druckverhältnissen durchgängig ist

Zur Klarung der Frage ob Endolymphe als Ursprungsort für die Perilymphe infrage kommt stellten wir Untersuchungen über die Durchgängigkeit der Reißnerschen Membran in Abhängigkeit von der Molekulgroße an Phosphor 32 Thymin in C[14] markierter Form und jodmarkiertes Humanalbumin wurde in den Perilymphraum ohne Druck eingetraufelt und die Endolymphe durch Kapillarität gewonnen Es ließ sich feststellen daß die Reißnersche Membran fur Jonen und kleine Aminosäuren durchgängig war während das Albumin mit einer Molekulgroße von 60 000 nicht mehr in die Endolymphe eindrang Wir sind uns bewußt daß wir durch die Eroffnung des Perilymphraumes unphysiologische Druckverhältnisse geschaffen haben Dennoch lassen die Versuche den Schluß zu, daß die Perilymphe kein direktes Filtrationsprodukt der Endolymphe sein kann Rauch (1960) hat Kalium und Natriumionen in markierter Form in den Perilymphraum gebracht und ebenfalls festgestellt daß die Reißnersche Membran fur Jonen durchgängig ist Das Kalium wandert hier sogar entgegen dem Konzentrationsgefalle offenbar unter Mitwirkung von Zellen in den Endolymphraum

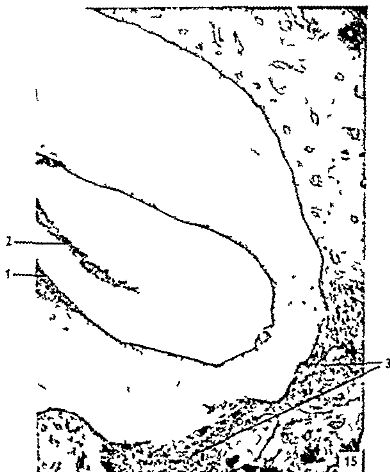



Abb. 15. Schnitt durch das Vestibulum mit Utriculus. Links außen ist die Macula utriculi (1) getroffen. Im Lumen Reste der Otolithenmembran (2). Am rechten und unteren Bildrand erkennt man in der knöchernen Labyrinthkapsel das Gefäßkonvolut (3)

sichere Methode, mit welcher eine Autoradiographie wasser- oder alkohollöslicher Stoffe durchführbar und damit eine Schwärzung der Transportwege der radioaktiv markierten Substanzen zu den Geweben möglich wäre. Bei der Fixation und Präparation der histologischen Schnitte für die Autoradiographie werden alle wasser- und alkohollöslichen sowie mit Formalin nicht fällbaren markierten Verbindungen aus dem Schnitt herausgelöst. Als Ausweg führten wir Untersuchungen mit tritiummarkierten Aminosäuren über den Eiweißstoffwechsel der Gewebe des Innenohres im Zeitstufenversuch durch. Für die Cochlea wurden ähnliche Versuche bereits von MEYER ZUM GOTTESBERGE (1961) beim Meerschweinchen beschrieben. Am Vestibularapparat wurden solche Untersuchungen noch nicht durch[illegible]hrt.

Wir injizierten die markierte Substanz intravenös und wählt[illegible] r-suchsdau[illegible]r 30 und 60 Minuten. Nach Gabe einer markierten [illegible] re wird diese [illegible]n unterschiedlicher Weise in das Eiweiß de[illegible] n [illegible]webe eing[illegible] MAURER (1959) spricht von der Um[illegible] r-

säure und versteht darunter diejenige Menge an markierter Aminosäure (AS), welche in 1 g Gewebe in einer Minute eingebaut wird. Es ist hierbei zu berücksichtigen, daß ein Einbau dieser markierten Aminosäure im wesentlichen nur in den ersten Stunden nach der Verabreichung stattfindet und dann ein rascher Abfall der spezifischen Aktivität der freien AS eintritt. Der Wiederabbau des markierten Eiweißes spielt in den ersten Stunden, wie Messungen der biologischen Halbwertszeiten verschiedener Gewebe von NIKLAS, QUINCKE, MAURER und NEYEN (1958) ergeben haben, noch keine Rolle. MEYER ZUM GOTTESBERGE (1961) hat festgestellt, daß schon 15 Minuten nach intravenöser Injektion markierter AS die wirkliche Umsatzrate derselben für die Gewebe der Cochlea erreicht ist. Unsere Untersuchungen haben eine Versuchsdauer von 30 und 60 Minuten, so daß die Ergebnisse die wirkliche Umsatzrate der AS für alle Gewebe des Innenohres aufzeigen, auch für diejenigen, welche durch Diffusion ernährt werden.

Bei unseren autoradiographischen Versuchen konnten wir feststellen, daß sich für verschiedene markierte AS ($H^3$ Phenylalanin und $H^3$ Lysin) Autoradiogramme (ARG) mit gleicher Schwärzungsverteilung über den Geweben des Innenohres fanden. SCHULTZE, OEHLERT und MAURER (1959) konnten dies auch für andere Gewebe des Organismus und KOBURG (1962) für Gewebe der Cochlea am Meerschweinchen feststellen. Nach Auffassung dieser Autoren ist dies ein Hinweis dafür, daß der Aminosäure-Inkorporation eine Synthese von Eiweiß zugrunde liegt und die Schwärzungsverteilung der ARG die Größe des Eiweißstoffwechsels in den verschiedenen Geweben wiedergibt.

Autoradiographische Untersuchungen von NIKLAS und OEHLERT (1956), NOVER und SCHULTZE (1960), OEHLERT und MAURER (1958) haben ergeben, daß die verschiedenen Gewebe bei der Maus, der Ratte und dem Kaninchen sehr unterschiedliche Eiweißumsatzraten haben. Zu der Gruppe der Zellen mit dem intensivsten Eiweißstoffwechsel im Organismus gehören nach Untersuchungen von KOBURG (1962) u. a. die Zellen von Drüsen mit eiweißhaltigem Sekret, die Ganglienzellen des zentralen und peripheren Nervensystems sowie die Zellen des Plexus chorioideus, des Ciliarkörpers und die Gefäßendothelien des RES.

Wie aus den Ergebnissen unserer Zeitstufenversuche zu ersehen ist, erkennt man sehr deutliche Unterschiede von Gewebe zu Gewebe hinsichtlich des Einbaues der aktiven Aminosäure. Bei einigen Geweben ist die Umsatzrate nach 30 und 60 Minuten Versuchsdauer der aktiven AS gleich, bei anderen steigt die AS-Umsatzrate nach 60 Minuten an. Unsere Untersuchungen bei der Cochlea des Kaninchens decken sich hierin mit den Untersuchungen von MEYER ZUM GOTTESBERGE (1960), welche dieser am Meerschweinchen durchführte.

Am Gleichgewichtsapparat ist festzustellen, daß es ebenfalls Gewebe gibt, bei denen nach 30 Minuten schon der optimale Einbau von AS erfolgt ist, während bei einer zweiten Gewebsgruppe die SK-Dichte nach 60 Minuten höher ist als nach einer kürzeren Versuchsdauer. Zur ersten Gruppe ge-

hören die Randzellen der Übergangszone und des Planum semilunatum der Crista ampullaris, sowie die Zellen des Gefäßbindegewebsnetzes in den Labyrinthwandungen, während zur zweiten Gruppe die Bogengangszellen, die Otolithenmembran und die Cupula zählen. Die Werte der AS-Umsatzrate entspricht bei der ersten Gruppe, welche NIKLAS und OEHLERT (1957), OEHLERT, SCHULTZE und MAURER (1959), NOVER und SCHULTZE (1960) beim Plexus chorioideus des Zentralnervensystems, dem Corpus ciliare der Aderhaut, sowie den Endothelien und Zellen des RES und schließlich bei den eiweißsezernierenden Zellen des Pankreas und der Nebennierenrinde fanden. Die Gruppe zwei zeigt eine SK-Dichte, wie sie von diesen Autoren bei der inneren Körnerschicht des Auges und der Basalzellenschicht der Zunge festgestellt wurden. Eine übersichtliche Darstellung der SK-Dichten dieser Gewebe gab KOBURG (1962).

Bei der Beurteilung der autoradiographischen Befunde des Vorhofbogengangapparates müssen zwei Ergebnisse im Hinblick auf die Problemstellung unserer Arbeit und für die Sekretion der Labyrinthflüssigkeiten besondere Beachtung finden: 1. Der Eiweißstoffwechsel in der Übergangszone und im Planum semilunatum hat die gleiche Größenordnung wie die Stria vascularis und 2. der Eiweißstoffwechsel in den Zellen des Gefäßbindegewebes des Labyrinthwandungen ist auffallend hoch.

Es ist schwer zu entscheiden, ob die Größe des Eiweißstoffwechsels einer Zelle oder eines Gewebe eine Aussage über die Funktion, insbesondere bei der Entstehung einer Körperflüssigkeit erlaubt. Dem Plexus chorioideus des Gehirns wird vorwiegend die Produktion des Liquors, dem Ciliarkörper, die Produktion des Kammerwassers mit einer möglichen aktiven Sekretion von Eiweiß zugeschrieben. Wenn diese Annahme stimmt, dann ist ein paralleler Schluß zu ähnlichen Organen im Innenohr ebenfalls gestattet. Die Hypothese, daß die Stria vascularis die Produktionsstätte der Endolymphe darstellt, wird durch unsere Untersuchungen bekräftigt. Auch MEYER ZUM GOTTESBERGE (1960) hat sich in diesem Sinne geäußert. Darüber hinaus glauben wir, daß auch die Übergangszone und das Planum semilunatum, welches die gleiche große SK-Dichte aufweist, wie die Stria vascularis, als sekretorisches Epithel für die Endolymphproduktion infrage kommt. Auch elektronenmikroskopische (BAIRATI und JURATO 1960) sowie fermenthistochemische Untersuchungen (NOMURA und BALOGH 1964) lassen darauf schließen, daß die erwähnten Gewebe eine sekretorische Funktion haben.

KOBURG und MEYER ZUM GOTTESBERGE (1960) fielen schon der hohe Eiweißgehalt im Ligamentum spirale oberhalb und unterhalb des Ansatzes der Reißnerschen Membran auf. Der Gewebsabschnitt oberhalb der Reißnerschen Membran deckt sich mit jenen Stellen, wo MYGIND (1945) und GRAF und PORITTI (1950) viele kleine Gefäße in einem weitmaschigen Bindegewebsnetz festgestellt haben.

Ein auffallend hoher Eiweißstoffwechsel war bei unseren Untersuch in der chlea noch im Tractus arteriosus et venosus des Modiolu im Li festzustellen

Im Bereich des Gleichgewichtsapparates ist ein ähnlich hoher Eiweißstoffwechsel im Gefäßbindegewebsnetz, welches den Labyrinthwandungen anliegt, zu erkennen

Alle erwähnten Gewebe in der Cochlea und im Gleichgewichtsapparat, welche einen hohen Eiweißstoffwechsel haben, weisen auch eine gute Blutversorgung auf Soweit diese Geweben nicht andere Funktionen erfüllen müssen, liegt der Schluß nahe, daß ihnen auch eine Beteiligung bei der Perilymphproduktion zukommt Aufgrund unserer Traceruntersuchungen konnen wir annehmen, daß die Perilymphe primär im Perilymphraum entsteht Wir glauben, daß die Bildungsstätte der Perilymphe in jenen Gebieten des Perilymphraumes zu suchen ist, in denen ein gefäßreiches Bindegewebsnetz vorliegt Das Gefäßbindegewebe des Ligamentum spirale oberhalb des Abganges der Reißnerschen Membran, dann der Tractus arteriosus et venosus des Modiolus sowie das Gefäßbindegewebsnetz im Gleichgewichtsapparat bietet sich dafur an Eine Resorption der Perilymphe in diesen Gewebsabschnitten erschiene dann ebenfalls naheliegend

In diesem Zusammenhang ist noch die starke Schwärzung der durch Diffusion ernährten Zellen der tympanalen Belegschicht bemerkenswert Ob diese Zellen an der Perilymphentstehung regulatorisch mitbeteiligt sind, kann zur Zeit noch nicht geklärt werden

Bei unseren Zeitstufenversuchen konnten wir feststellen daß die Sk-Dichte über der Basilarmembran nur sehr gering war Sie lag teilweise kaum uber dem Nullwert und liegt auf jedem Fall unter der Sk-Dichte der tympanalen Belegschicht Ein Antransport von AS aus der Scala tympani über die tympanale Belegschicht und Basilarmembran in die Raume der Cortilymphe scheidet unserer Meinung nach aus

# IV. SCHLUßFOLGERUNGEN

1 Die Perilymphe wird im Perilymphraum selbst gebildet Hierfür sprechen Traceruntersuchungen mit markierten Stoffen, wie Phosphor 32 und Jod-markierten Albuminen Außerdem beweisen vergleichende analytische Untersuchungen hinsichtlich des Elektrolytgehaltes, der freien Aminosäuren und der Proteine, daß Unterschiede in der chemischen Zusammensetzung zwischen Perilymphe und Liquor bestehen müssen Aufgrund der histologischen und autoradiographischen Untersuchungen kommen als Produktionsstätten die Gefäße des Tractus arteriosus et venosus im Modiolus der Schnecke und kleine Gefäße in der Scala vestibuli, die oberhalb des Abganges der Reißnerschen Membran liegen, in Betracht Im Gleichgewichtsapparat konnten plexusartige Gefäße im subperiostalen Bindegewebe der Labyrinthwandungen diese Funktion übernehmen

2 Der Aquaeductus cochleae ist für Ionen und Stoffe bis zu einer Molekulgröße der Albumine (60 000) in Richtung Subarachnoidalraum-Perilymphraum durchgängig Für Stoffe in der Größenordnung lichtmikroskopischer Sichtbarkeit, wie etwa Mitochondrien, besteht keine Durchlässigkeit Es scheint möglich, daß dem Liquor im Subarachnoidalraum in Notfällen und bei unphysiologischen Bedingungen die Rolle eines Ergänzungsreservoires zukäme

3 Versuche bei einseitigem Verschluß des Aquaeductus cochleae zeigten daß Liquor über andere Wege als den Aquaeductus cochleae in den Perilymphraum eindringen kann Wahrscheinlich kommen dafür die perineuralen Lymphscheiden infrage Ein Nachströmen von Liquor in den Perilymphraum wurde nur unter unphysiologischen Bedingungen beobachtet

4 Ein Stoffaustausch von Ionen und Aminosäuren aus dem Perilymph- in den Subarachnoidalraum ist möglich Dieser Stoffaustausch ist aber nur niedrig

5 Es findet ein schneller Stoffaustausch für Ionen zwischen den Perilymphräumen des linken und rechten Ohres statt

6 Die Reißnersche Membran erweist sich als durchgängig für Ionen und Aminosäuren nicht aber für Moleküle in der Größenordnung der Albumine

7 Die Perilymphe wird im Perilymphraum resorbiert Hierfür sprechen Versuche bei denen radioaktiv markierte Substanzen in den Perilymphraum gebracht wurden und diese schon nach kurzer Zeit im peripheren Blut nachweisbar waren

8 Autoradiographische Untersuchungen am Gleichgewichtsorgan mit [illegible] markierten Stoffen ergaben Hinweise, daß auch die Randzellen der

Crista ampullaris (Übergangszone und Planum semilunatum) als Produktionsstätten der Endolymphe anzusehen sind.

9. Eine Abstammung der Corti-Lymphe aus der Perilymphe der Scala tympani ist nicht wahrscheinlich. Bei autoradiographischen Zeitstufenversuchen zeigte sich, daß die Zellen der tympanalen Belegschicht konstant eine höhere Silberkorndichte aufwiesen als die Basilarmembran.

# V. ZUSAMMENFASSUNG

Zur Klärung des noch umstrittenen Problems der Herkunft der Perilymphe wurden experimentelle Untersuchungen am Kaninchen durchgeführt

Für unsere Problemstellung hielten wir Traceruntersuchungen mit radioaktiven Stoffen, wie Phosphor$^{32}$, C$^{14}$-markierten Aminosäuren, Phosphor-markierten Zellgranula und Jod$^{131}$-markierten Albuminen am geeignetsten Die radioaktiven Substanzen wurden intravenös, subokzipital und in den Perilymphraum selbst appliziert Im Zeitstufenversuch von 5 Minuten bis zu mehreren Tagen wurden stets gleiche Mengen an Perilymphe, Liquor und Blutserum entnommen und die Radioaktivität gemessen Der Vergleich der Radioaktivitäten in den einzelnen Körperflüssigkeiten gestattete Rückschlüsse über den Antransport der aktiven Substanzen zu den jeweiligen Körperflüssigkeiten

Ergänzt wurden diese Experimente durch autoradiographische Untersuchungen mit tritiummarkierten Aminosäuren an der Cochlea und am Gleichgewichtsorgan

Daneben wurden noch vergleichende chemisch-analytische Untersuchungen der Elektrolyte, der freien Aminosäuren und Proteine in Perilymphe, Liquor und Blut durchgeführt

Voraussetzung für die Durchführung dieser Versuche war, die physiologischen Verhältnisse im Labyrinth, wie z B die ausbalancierten Druckverhältnisse, zu wahren und die Perilymphe ohne Blutbeimengung zu gewinnen Zur Lösung dieses Problems wurden entsprechende Operationsmethoden ausgearbeitet, es wurde auch die Präparation des Innenohres in einer modifizierten Operationstiefkühltruhe (Kryostat) angegeben

Eine Reihe weiterer technischer Probleme wie die Messung dieser kleinsten Mengen an Labyrinthflüssigkeiten, mußte gelöst werden

Die erzielten Resultate wurden in 9 Schlußfolgerungen dargelegt

Aus unseren Untersuchungen geht hervor, daß die Perilymphe im Perilymphraum selbst entstehen muß Die Perilymphe stellt keine stagnierende Flüssigkeit dar, welche nur als Wasserkissen für den Endolymphschlauch dient, sondern unterliegt einem regen Wechsel also einer ständigen Erneuerung und Resorption Die Stoffwechselbeziehungen zum Liquor des Subarachnoidalraumes und zu den anderen Labyrinthflüssigkeiten sind sehr vielfach

Herrn Prof Dr W Büngeler, dem Direktor des Pathol Institutes der Universität München und Herrn Prof Dr Dr H Wrba jetziger Director des Instituts für experimentelle Medizin in Heidelberg sei an dieser stelle dafür gedankt daß es mir erlaubt wurde die Einrichtungen des Institutes und insbesondere des Isotopenlabors zu benutzen Herrn Prof Dr Dr Wrba habe ich noch besonders zu danken für seine Anregungen und Unterstützungen bei den Tranceruntersuchungen

Herrn Prof Dr W Maurer Leiter des Institutes für medizinische Isotopenforschung der Universität Köln danke ich für die freundliche Hilfe und für wertvolle Hinweise bei der Durchführung der autoradiographischen Untersuchungen

# VI. CONCLUSIONS

1. Tracer studies with radioactive substances—Phosphor-32—and jodinated albumines *indicate* that the *perilymphe* is produced within the perilymphatic space. Furthermore comparative studies concerning electrolytes, free amino acids and protein demonstrate certain differences of the chemical compounds of the perilymphatic and cerebrospinal fluid. According to histological and autoradiographical *findings* the vessels of tracies arteriosus and venosus in the cochlear modiolus and the small vessels of the scala vestibuli, just above the origin of the Reißner's membrane seem to be the secretory places. In the vestibular organ the plexuslike vessels in the subperiostal connective tissue of the labyrinthal wall are able to take over this secretory function.

2. The cochlear *aquaeduct* is permeable for Ions and substances up to the molecular size of the albumins (60 000) in direction to the subarachnoidal-perilymphatic space. There exists *no* permeability for substances of the size of light-microscopical visibility like *mitochondria*. It seems possible that the cerebrospinal fluid of the subarachnoidal space has a certain reserve function in cases of emergency and under unphysiological conditions.

3. Studies with and unilateral obstruction of the cochlear *aquaeduct* showed, that the cerebrospinal fluid can use other pathways than the cochlear aquaeduct to penetrate into the perilymphatic space. Probably the perineural lymphatics can act as a pathway. A flow of cerebrospinal fluid into the perilymphatic space could only be observed under unphysiological conditions.

4. An exchange of Ions and amino *acids* from the perilymphatic to the subarachnoidal space is possible. This exchange however is *quantitatively* very low.

5. There is a rapid exchange of Ions between the perilymphatic spaces of the left *and* right ear.

6. The Reißner's membrane proved to be *permeable for* Ions and amino acids, but not *for* molecules of albumine-size.

7. *After* incorporation of radioactive substances into the perilymphatic space these substances could be traced in the peripheral blood within a short time. This indicates that the perilymph is resorbed in the perilymphatic space.

8. Autoradiographical studies of the vestibular organs with tritium-labelled substances indicated, that also the marginal cells of the crista ampullaris (transition zone and planum semilunatum) have to be considered as secretory places of the endolymphatic fluid.

9 An origin of the lymphatic fluid of the organ of Corti from the perilymphatic fluid of the scala tympani is not very likely Autoradiographic studies at different times showed constantly a higher incorporation of silber granula in the epithelial coverage of the scala tympani than in the basilar membrane

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# BONE CONDUCTION

## STUDIES IN EXPERIMENTAL ANIMALS

*A collection of seven papers*

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# PREFACE

The present collection of seven papers deals with various experiments on bone conduction in laboratory animals These experiments were conducted over a period of nearly five years The work was started in 1960 in the research laboratories of the Department of Otolaryngology of the University of Iowa and, after 1962 continued in the research laboratories of the Department of Otolaryngology, College of Physicians and Surgeons, of Columbia University The experiments were brought to conclusion late in 1964

The experiments grew out of discussions between Dr J R Tabor, then a resident in Otolaryngology at Iowa, and myself Some preliminary experiments were reported (J Tonndorf and J R Tabor Closure of the Cochlear Windows Its Effect upon Air- and Bone Conduction *Ann Otolaryngology, 71* 1–25 (1962)) This was followed by a separate study in cochlear models (J Tonndorf Compressional Bone Conduction in Cochlear Models, *J Acoust Soc Am*, *34* 1127–1131 (1962)) Of the present experiments only one paper summarizing the most obvious clinical conclusions has been published (J Tonndorf Animal Experiments in Bone Conduction Clinical Conclusions *Ann Otolaryng 73* 659–688 (1964)) As the experiments progressed it became apparent that the phenomenon of bone conduction is quite complex and that details, as they were collected, had to tie in with others to be explored later

Many papers have been published on the subject of bone conduction, but their results vary to quite some extent Usually, in such cases the investigative techniques employed are different and their results should not be compared Much time and effort, therefore, was spent in developing and validating experimental techniques More often than not, this period of development took considerably more time than the execution of the particular series of experiments evolving from it

Presentation in a series of quasi independent papers was chosen for several reasons (1) During the long period covered a number of co-workers participated in these studies, each one on one or several sub subjects In a single monograph it would have been difficult to identify everyone with his particular contribution (2) A chronological write up preserves the gradual development of concepts as they evolved from the various phases of study Frequent cross references are made in each paper to findings in later studies and occasionally, later material is used in support of conclusions from earlier data However, some conclusions, even when incomplete or faulty, are reported in earlier papers They are not supplemented and or corrected until later, because too much additional

# SYNOPSIS

# A REVISED THEORY OF BONE CONDUCTION

JUERGEN TONNDORF

The currently accepted theory of Bone Conduction has a history dating back for more than 100 years Quite a few of the older concepts still have validity today The literature is quite voluminous, even when one disregards purely speculative dissertations Excellent recent summaries of the current status are those of Hood (*J Acoust Soc Am*, *34* 1325–1332 (1962)) and of Naunton (Chapter I in *Modern Development in Audiology*, J Jerger, ed, Academic Press, New York, 1963) However, with some noted exceptions, many of the earlier writings were based upon little experimental evidence Quite often, authors founded rather elaborate concepts upon casual clinical observations of specific, but limited kinds Also, the laudable quest for simplifying generalizations led to attempts to find a single, unifying principle underlying the phenomenon of bone conduction A good example of this is the heated debate carried on at the end of the last century, on "osseous" versus "osseo tympanic" bone conduction which in some manner has continued till today

The series of experiments to be reported in the following seven papers was conducted in laboratory animals, mainly in cats However, rats, guinea pigs, and dogs were also used on occasion, primarily in an effort to identify species-specific differences One study was conducted in cadaver heads of cats In living animals, response indication was by registration of cochlear microphonic responses The bone input was monitored by an accelerometer Analytic approaches were used, i e the function of the ear under test was altered in a variety of steps designed to affect various structures of the ear which, in earlier publications, had been suggested to contribute to the over-all bone conduction response

Thus, most of the current experiments started out from the results of and the hypotheses developed by earlier writers, notably those of Herzog & Krainz, Békésy, E Barany, Groen, Kirikae, E H Huizing, and others Some other concepts were developed as the work progressed Due to the large number of past writings on the subject only a few really novel concepts can be claimed to have been generated by the present series of experiments However, much of what has been written before on subjects (such as individual bone-conduction components and their relative importance, con tributions due to energy travelling through the interior of the skull, the very existence of compressional bone-conduction, and many others) was either lacking in quantitative support or was based upon circumstantial

TABLE 1 *Mechanisms of Bone Conduction Responses.*

| Mode | Effect upon | Modified by |
|---|---|---|
| I Energy radiated into external canal | Tympanic membrane | (a) Outer canal opening<br>(b) canal resonances<br>(c) impedance of tympanic membrane |
| II Acceleration of temporal bone | A Ossicular chain ("ossicular inertia") | (a) Impedance of ossicular chain,<br>(b) air column in external canal<br>(c) air enclosed in middle ear |
| | B Inner ear fluids (inertial effect) | (a) Impedance of cochlear windows & of cochlear partition |
| III Distortional vibrations of temporal bone | Cochlear shell ("pure compressional component") | (a) Round window release<br>(b) oval window release,<br>(c) "third" window release |

evidence It is felt that the present experiments have substantiated a number of such concepts while repudiating others

The following *revised concept of bone conduction* has evolved From the point of contact of vibrator and head, vibrations travel (a) as *surface waves* along the skin and soft tissues, (b) as *transversal waves* by way of the bones of the skull, and (c) as *pressure* and/or *translation waves* through the interior of the skull Of these, the surface waves along the soft tissues are probably of little, if any, importance The other two upon reaching the temporal bone, interfere with each other, i e they combine, depending upon their amplitude and phase relationships Part of the pressure waves of the skull interior acts directly upon the cochlea through the so-called 'third window", the sum of the communications of the cochlea with the endocranial spaces

The bone conduction response of the ear itself is brought about by three basic modes

(a) *the reception of sound energy radiated into the external ear canal,*
(b) *the inertial response of the middle-ear ossicles and the inner-ear fluids*, and
(c) *the compressional response of the inner ear spaces*[1]

[1] According to the present findings (for details cf paper No I) the term 'compressional bone conduction" is really not appropriate any more A *distortion* of the cochlear shell does not necessarily entail a *compression* of the enclosed spaces as the classical theory had it The finding that inner ear responses to bone conduction stimulation function in the absence of any cochlear openings (J Tonndorf, *J Acoust Soc Am* 34 1127 1131 (1962)) attested to this fact Nevertheless the present writer realizes [illegible] be very difficult if not impossible to discard a term as firmly accepted as [illegible] here and therefore refrains from suggesting a new one

In classical terminology, the first two may be considered *osseo-tympanic* modes, the last one the *osseous* mode

(a) *Radiated sound energy*—This energy is given off by the walls of the external canal and is received by the tympanic membrane Its reception is modified by the outer opening of the external canal acting as a high-pass filter and by any impairment of transmission through the middle ear

(b) *The inertial response*—This response is due to (i) the middle-ear ossicles and (ii) the inner ear fluids The response of the middle-ear ossicles is modified (aa) by the resonant properties of the air column within the external canal and (bb) by those of the air enclosed in the middle ear Both of these act as loads upon the tympanic membrane and are minimal at their respective resonant points In addition, the latter load also acts upon the round window The inertial response of the inner ear fluids interacts with the compressional response

(c) *The compressional response*[1]—The compressional response of the inner ear spaces is due to distortional vibrations of their shell, and it is independent of any cochlear openings the 'pure compressional component" It is modified by the "three' cochlear windows That is to say, it is reinforced by the yielding of the round window, while the oval window acts as a shunt Of the "third cochlear window", only the effect of the cochlear aqueduct was assessed Its action augments that of the round window, but only in the low-frequency range

The response of the normal ear *integrates* all three modes and the various modifying components, according to their amplitude and phase relationships However, when parts of the ear are missing and/or functionally eliminated, the total bone conduction response is altered accordingly

The present concept is summarized in Table 1

# I QUANTITATIVE EVALUATION OF BONE CONDUCTION COMPONENTS IN CATS

JUERGEN TONNDORF, RICHARD A CAMPBELL
LESLIE BERNSTEIN AND JOHN P RENEAU

## Summary

Changes in amplitude and phase of cochlear microphonic responses to bone conducted signals were recorded following various alterations of the middle-ear and cochlear structures Seven different *components* which *contribute in varying degrees to the total* bone conduction *response* were identified and subsequently isolated by calculation These components are (1) middle ear (ossicular) inertia (2) *middle ear cavity* compliance (3) inner ear compression (4) round *window* pressure release (5) oval window *pressure* release (6) inner ear fluid inertia and (7) pressure release through the cochlear aqueduct Although the existence of the latter component gave evidence as to the reality of the third window effect (the sum total of both aqueducts and all perivascular and perineural channels through the cochlear capsule) this latter effect has not been assessed in toto

## A Introduction

The response of the cochlea to bone conducted vibrations, in the opinion of most of today s writers results from the combined effect of a variety of factors or *bone conduction components*

The factor *of inner ear compression* was first postulated by *Rejto* (1914) and became generally recognized after the model experiments *of* Herzog & Krainz (1926) The concept of *middle ear ossicular inertia* was originally established by Barany (1938) *on the* basis of *experiments* in human subjects The effect of *inner ear fluid inertia* was deduced by Wever & Lawrence (1954) based upon considerations similar to those of Baranys Additional factors have been proposed by various writers *However*, as stated above there is almost universal agreement that such modes as compressional or innertial bone conduction for example are not exclusive entities but rather that the ultimate cochlear *response represents* the integrated effect of *all* of these components Earlier at the *end* of the 19th Century there had been two strictly separate schools of thought

Th s w rk was m a le possible through the support b the following grants Research r Nl I O lar ngology training grants NB o11C Audiology training grants
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One maintained that the vibratory energy would reach the cochlea exclusively by a *direct* ("osseous") route The other thought in terms of an *indirect* ("osseo tympanic") one Allen & Fernandez (1960) might be considered modern proponents of the first school and Wever & Lawrence (1954) of the second one, although none of them, apparently, think in terms of exclusive modes any longer, but rather of dominant ones

The two principal modes, compressional and ossicular-inertial, will be briefly outlined here

In the *compressional mode*, according to Herzog & Krainz (1926), the vibratory energy reaching the cochlea was thought to bring about alternate compressions and expansions of the cochlear capsule At the low amplitudes involved the cochlear fluids had to be regarded as incompressible Therefore, the volume changes of the cochlear spaces due to the compressions and expansions of their walls required some compensatory mechanism This mechanism was assumed to be given by the yielding of the cochlear windows However, it was realized that such a mechanism would not necessarily produce the differential action between *scala vestibuli* and *scala tympani* required for the displacement of the cochlear partition This action in the opinion of Herzog & Krainz, might be brought about by two independent mechanisms

(a) The oval window is known to be less compliant than the round window [according to more recent findings of Kirikae (1960) the compliance ratio is 1 20] so that on contraction of the cochlear capsule the excess in fluid displacement on the vestibular side must force the partition downward, toward *scala tympani*

(b) The surface of the contracting walls on the vestibular side is known to be larger than that on the tympanic side (The ratio of the two volumes is usually given as 5 3, thus, that of the areas is approximately 3 2) Consequently, more fluid is displaced in *scala vestibuli* than in *scala tympani* forcing the cochlear partition down even further than under condition (a) It is noted that both of these mechanisms operate in phase with each other

According to Barany's concept of the *ossicular inertial mode* the two major ossicles, malleus and incus, vibrate together as one body, i e there is no movement in the incudo-maller joint (a fact recently confirmed by Møller, 1963) The ossicles have but one degree of freedom and rotate around an axis running from the anterior mallar ligament to the incudal ligament roughly in an anteroposterior direction The center of gravity of the two ossicles lies very close, but not quite upon, the axis of rotation making their moment of inertia in response to translatory motion rather low When the skull undergoes forced vibrations (especially from side to side) the ossicles participate in this motion, but because of their loose coupling and of their different resonant properties, at amplitudes and phases different from those of the skull Thus, a relative motion is set up between the stapes (which is part of the ossicular chain) and the oval

# I. QUANTITATIVE EVALUATION OF BONE CONDUCTION COMPONENTS IN CATS

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## Summary

Changes in amplitude and phase of cochlear microphonic responses to bone-conducted signals were recorded following various alterations of the middle-ear and cochlear structures. Seven different components which contribute in varying degrees to the total bone conduction response were identified and subsequently isolated by calculation. These components are (1) middle-ear (ossicular) inertia, (2) middle-ear cavity compliance, (3) inner-ear compression, (4) round window pressure release, (5) oval window pressure release, (6) inner-ear fluid inertia, and (7) pressure release through the cochlear aqueduct. Although the existence of the latter component gave evidence as to the reality of the "third window effect" (the sum total of both aqueducts and all perivascular and perineural channels through the cochlear capsule), this latter effect has not been assessed *in toto*.


## A. Introduction

The response of the cochlea to bone-conducted vibrations, in the opinion of most of today's writers, results from the combined effect of a variety of factors or *bone-conduction components*.

The factor of *inner-ear compression* was first postulated by Reijö (1914) and became generally recognized after the model experiments of Herzog & Krainz (1926). The concept of *middle-ear ossicular inertia* was originally established by Barany (1938) on the basis of experiments in human subjects. The effect of *inner-ear fluid inertia* was deduced by Wever & Lawrence (1954) based upon considerations similar to those of Barany. Additional factors have been proposed by various writers. However, as stated above, there is almost universal agreement that such modes as "compressional" or "inertial" bone conduction, for example, are not exclusive entities but rather that the ultimate cochlear response represents the integrated effect of *all* of these components. Earlier, at the end of the 19th Century, there had been two strictly separate schools of thought.

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[illegible]

## B Method and Procedure

In general, the experimental procedure was identical to that of the earlier study (Tonndorf & Tabor, 1962) Again, young cats were used (weight 1 5 to 2 5 kg) Cochlear microphonics were recorded from the basal turn by means of differential electrodes which were sealed in fluid-tight by means of dental cement An indifferent electrode was placed into the neck muscles Before the cement was applied, however, silicon of high viscosity (100,000 centistokes) was deposited under pressure around the intra-cochlear electrodes in order to protect the cochlear fluids and tissues against the acidity of fresh dental cement (Fig 1) This procedure had the added advantage of preventing air from being sealed in under the cement cover Even small air bubbles, when brought into the cochlea, will act as compressible elements in an otherwise incompressible medium—the cochlear fluids The transparent silicon allowed detection of very small air bubbles which could be removed before the cement cover was applied Essentially the same technique was used for the occlusion of the cochlear windows (Fig 2) As a rule, "Grip" dental cement (Caulk and Co ) was given preference over ordinary zinc-oxide cements because when properly applied with its liquid primer, it provides an excellent bond to the underlying bone Zinc-oxide cements were only used for temporary occlusions, for example the occlusion of the round window In that case, several undercuts were made into

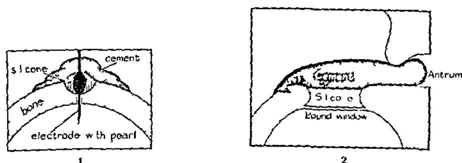



Fig 1 Intra-cochlear, sealed in electrode (schematic) Note the slight indentation made into the bone on the bottom of which there is the actual small perforation After its introduction the electrode is first covered by silicone (100 000 centistokes) before the cement cap is applied It is advisable to lay down a low wall of cement around the indentation before perforation i e as long as the bone can be kept completely dry This will aid the procedure later on (from Tonndorf & Tabor 1962)

Fig 2 Occlusion of the round window (schematic) The niche is completely filled with silicone and all air bubbles are carefully removed before the cement cap is applied The latter is held firmly in place with the aid of several undercuts made superiorly anteriorly and inferiorly into the promontory and posteriorly by wedging the cement into the small air cell which corresponds to the human antrum (from Tonndorf & Tabor 1962)

the bone to provide a firm hold for the cement cap (Fig 2) Further experimental details were described in the earlier study (Tonndorf & Tabor, 1962)

A change in anesthesia was made which is worth reporting In long-lasting experiments of 9–12 hour duration, the respiratory-depressant effect of nembutal became quite apparent Support by artificial respiration was tried for a while by means of a small-animal respirator (Phipps and Bird) However, the equipment proved to be too noisy for auditory testing so that it had to be turned off during each testing period A superior solution was provided by premedication with a tranquilizing agent, a method which, according to Dolowy *et al* (1959), reduces the need for barbiturates By trial and error, the following dosage was worked out. Chlorpromazine, i m, 14 mg/kg wt, followed fifteen minutes later by sodium nembutal intra-thoracically, 22 mg/kg wt This is about two-thirds of the dose used previously without premedication Furthermore, the chlorpromazine made the need for supplemental injections less frequent so that an experiment of 10 hours duration could be carried out with a total nembutal dose of 40 to 45 mg/kg wt, compared to 60 to 70 mg/kg wt, used previously. Animals breathed freely and deeply for the duration of the experiment, and there were no untoward effects, provided the body temperature was kept normal Tranquilizers as basic anesthetics are known to lower body temperature Therefore, the temperature was maintained with the aid of a water-circulating heating pad (Gorman-Rupp & Co) which is free from 60 Hz interference

The test situation was quite rigidly controlled After its initial preparation, the animal was placed on a board and put in a testroom which was electrically and acoustically shielded Its head, with the test ear facing upward, was held loosely in a headholder and rested upon a soft rubber pad The position of the head was never changed during the entire test run The bone vibrator (Western Electric D-80904) rode up and down in a rigid sleeve and made contact with the animal's head under its own weight Thus, it was always applied with the same pressure The contact rod of the vibrator was equipped with a sharp tip which was received in a small dimple, fashioned from dental cement and located at the juncture of the inferior temporal line, the mastoid ridge, and the nuchal line, an easily identifiable landmark The dental cement had the added advantage of providing electrical insulation between the head and the vibrator Such insulation was found necessary in order to avoid ground loops

With respect to *amplitude* measurements, the above-mentioned method gave good reproducibility However, it was found that reliable *phase* measurements of the microphonic responses, relative to the electrical input signal were considerably more difficult to obtain than amplitude measurements The difficulty arose from the need to remove the animal on its board from the test chamber between measurements for proper execution of the experimental alterations of the ear This necessitated breaking the

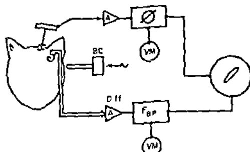



Fig 3 Testing arrangement, schematic BC bone-conduction vibrator, Diff A differential amplifier (Tektronix 122 modified for an 80 dB gain), $F_{BP}$ band pass filter (Allison 2A), VM vacuum tube voltmeter, A amplifier
The vibration pick up (Brüel & Kjaer 4303) the phase shifter (Grason Stadtler E3520B), and the oscilloscope (Tektronix 532) are shown by appropriate pictorial symbols For further explanation cf text

contact between the bone-conduction vibrator and the skull which could not be restored in exactly the same manner Consequently, phase measurements varied in an unpredictable way, although care had been taken to restore the animal board each time in the same position so that presumably the head and the vibrator were reconnected as before

It thus became necessary to monitor the mechanical input to the skull To this end, a small rectangular extra dural trephine opening was made into the middle fossa, just above the tentorium and close to the external canal A ⊥-shaped screw was slipped into this opening and secured tightly to the bone by means of a nut and washer Screwed onto this rather solid connector was a small vibration pickup (Brüel & Kjaer, Type 4303), the output of which served as reference for all measurements Admittedly its location was about 12 mm away from the point of contact with the vibrator and therefore phase readings obtained here were not necessarily representative of those at the bone conduction input However, errors introduced in this manner remained constant for all experimental conditions and thus did not affect the registration of *relative* changes

Fig 3, in a schematic manner, shows the experimental testing arrangement The stimulus signal (seven frequencies in octave or near octave steps between 150 Hz and 7500 Hz) was applied via the vibrator Response amplitudes were read through a band pass filter (Allison 2B) in the cochlear response line in reference to the vibration pickup Noise levels were slightly below 1 $\mu$v, varying somewhat with individual filterbands

For phase readings, the phase shifter in the monitor line was adjusted so that both signals appeared precisely in the same phase at the oscilloscope ($\Delta\varphi = 0°$), and the required value was recorded from the phase shifter Simple consideration will show that any corrective adjustment of the phase shifter, necessitated by a given alteration in the animal's ear, corresponded in magnitude and in sign to the phase shift of the microphonic response

induced by the experimental alteration in question Reliability in test retests was approximately $\pm 5°$ In other words phase shifts of 10° to 15° between different tests represented meaningful changes

Amplitude readings were obtained in the following manner first the background noise level was noted for a given filter band Then the appropriate signal was introduced and adjusted in amplitude in order to give a response 3 dB above the noise level It is easy to show (Tonndorf 1951) that in this case signal and noise are of equal voltage Since all noise levels per filterbands were less than 1 $\mu$V readings were obtained at very low levels i e well within the linear portion of the input/output functions

Amplitude readings were obtained once and phase readings three times for the series of seven frequencies in each test situation That is to say after the phase readings had been taken for all seven frequencies the vibrator was lifted off and was immediately replaced Thereafter all seven measurements were taken once more and then the procedure was repeated a third time The average of all three tests was taken as representative for each frequency Occasionally large deviations were found at 500 Hz 1000 Hz, and to a lesser degree at 2000 Hz An explanation for such occurrences was found in a later study (cf paper No II), when the effect of eardrum loading was measured It turned out that at the three frequencies mentioned especially at 500 Hz and 1000 Hz very small loads produced large phase changes It is conceivable therefore that the occasional aberrant readings were caused by seepage of blood or of tissue fluids onto the tympanic membrane

Care was taken that the acoustic output of the vibrator and its electromagnetic field did not affect the recorded responses (The vibrator was shielded in a mu metal cover ) For all test frequencies such interference did not occur until signals levels were reached several orders of magnitude above those actually used

Measurements were taken in a variety of experimental situations a list of which follows

(1) Closure of the external osseous canal
(2) Stepwise amputation of the middle ear
    (a) disarticulation of the incudostapedial joint
    (b) severance of the stapedial tendon
    (c) removal of the stapedial superstructure
(3) Stapedectomy
(4) Window occlusion
    (a) round window
    (b) oval window
    (c) both windows simultaneously

I ch individual result for a given frequency was referred to that obt... f r the same frequency in the normal situation i e with the ... t et and the bulla resealed Many of the tests listed above were

TABLE 1 *List of Procedures*

R – reference tests

| | | |
|---|---|---|
| R | (1) Normal animal | (a) Bulla open |
| | (2) Round window occluded | |
| | (3) Round window reopened | (b) Bulla sealed |
| (R) | (4) Incudo stapedial joint disarticulated | |
| | (5) Stapedial tendon cut | |
| R | (6) Stapedial superstructure removed | |
| | (7) Round window occluded | |
| | (8) Both windows occluded | |
| | (9) Round window reopened (oval window occluded) | |

also performed with the bulla open, and a separate series of all tests was run with the cochlear aqueduct closed

Results from a total of 20 animals were finally evaluated Ten more animals had served to establish the general procedure It was, of course, not possible to carry out the entire program in each animal Therefore, schedules varied somewhat, although each of them was followed for a series of five to six animals A sample schedule is given in Table 1

Results sometimes varied considerably between animals It is necessary, therefore, to first describe the method of *data handling*, before *average* results can be presented

## C Variation Between Animals

First of all, an erroneous variation may be introduced by the conventional method of plotting phase data such as that used, for example, in Fig 5, bottom Values for a given frequency are occasionally distributed around the two opposing ends of the graph, i e close to the +180° and −180° points This misleading presentation was avoided by the use of polar graph paper, with phase as the angular and continuous co ordinate, so that values around ±180° were properly lumped together

It was soon noted that for a given frequency there was some interdependence between the magnitude of the observed amplitude changes and the associated phase shifts for any group of animals subjected to the same tests Moreover, small amplitude changes sometimes varied between positive and negative values Frequently in such cases, positive amplitude changes were associated with phase shifts which differed by approximately 180° from those accompanying negative amplitude changes

It was felt, therefore, that a separate evaluation of amplitude and phase shifts might give a wrong impression of between-animal variations It was decided to combine both data into a single figure expressing the above mentioned correlations To this end the changes observed for a given frequency and in a given experimental situation, usually expressed *re*

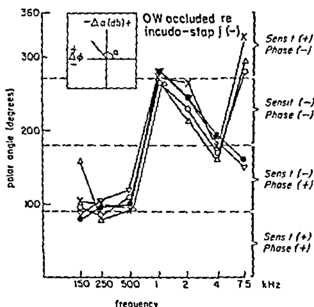



FIG. 4. Polar angles for seven signal frequencies in five animals computed from the following test: Oval window occluded re interruption of the incudo-stapedial joint. The insert shows the derivation of the polar angle. Note the labeling of the four quadrants on the right hand side of the main graph.

normal ear, were plotted in a rectangular co-ordinate system: $\Delta a$ along the horizontal axis and $\Delta\varphi$ along the vertical axis. When the resultant point was connected with the origin, in the manner shown in the insert of Fig. 4, a polar angle $\alpha$ was obtained. Depending upon the signs of both changes, this angle could have any value between 0° and 360°. The results were then entered into a plot of polar angle *vs* frequency (main graph of Fig. 4). In this manner of plotting, the original information was not entirely lost, for the labeling of the four quadrants on the right-hand side of the graph still conveyed a gross classification of phase and amplitude changes. As the example of Fig. 4 shows, the angle $\alpha$ displayed but small variations between animals, whereas amplitude and phase data taken separately showed much larger variations. Especially noteworthy in Fig. 4 is the data distribution for 150 Hz, 250 Hz, 1000 Hz, and 4000 Hz. At each of these frequencies, individual data clustered around a boundary between quadrants separating either positive and negative amplitude changes (at 90° and 270°) or such phase shifts (at 0° and 180°). Separate evaluation of amplitude and phase changes simply could not have made any sense in these instances. Yet in the manner displayed here, values at each frequency are clearly shown to belong to the same group. The situation at 7500 Hz illustrates the other point made above. At this frequency, there is a clear separation of values into two distinct groups. In this particular case, increased sensitivity went with negative phase shifts and vice versa. Closer inspection will show that one group is just the opposite of the other. That

is to say, inversion of the signs for both amplitude and phase changes of the members of one group would actually bring them into close association with those of the other group

The polar angles were utilized for the derivation of *average* amplitude and phase shifts First, a mean polar angle was formed by averaging individual values (In cases like that of 7500 Hz of Fig 4, the values of the smaller group were changed by 180° ) Thereafter, the length of each individual vector, $\Delta a$ vs $\Delta\varphi$, was projected upon the terminal side of the mean polar angle (or in cases like that of 7500 Hz in Fig 4, upon its projection into the opposite quadrant) Finally, average phase and amplitude values were obtained by averaging the projected points along the terminal side of the mean polar angle and reading the resultant in terms of $\Delta a$ and $\Delta\varphi$ All average values cited in the present paper have been obtained in this manner

## D Results

### 1 *Opening of the Tympanic Bulla*

When the tympanic bulla was opened (Fig 5) there were small amplitude losses in the high-frequency region and still smaller gains in the middle frequencies Comparable observations had been made by Benson & Eldredge (1955) with respect to air-conducted signals According to Békésy (1936), the compliance of the tympanic membrane is mainly due to the air enclosed in the middle ear Consequently, opening the tympanic bulla must eliminate this compliance factor Groen (1962) had calculated this factor with respect to the bone-conduction response in man

### 2 *Sealing the Osseous Ear Canal*

In sealing the short osseous ear canal care was taken to eliminate all air pockets in front of the tympanic membrane Thus, this procedure amounted essentially to a fixation of the latter membrane and presumably of the ossicular chain as well Møller (1963) had found no displacement in the incudo-mallar joint in response to air-conducted signals in the audiofrequency range In the present experiment, the *amplitude losses* due to the fixation of the tympanic membrane (Fig 5) and those due to stapedial fixation (Fig 6) were fairly similar to each other, if not identical However, the accompanying phase shifts in the two cases were clearly of opposite tendencies Since a firm stapedial fixation practically eliminates the effect of ossicular inertia, it appears that the fixation of the tympanic membrane does not completely immobilize the ossicular chain, and thus permits some, although probably minor, displacements These might be due to either (a) the fact that not all of the tympanic membrane is tightly coupled to the ossicular chain, or (b) some yielding of the incudo-mallar joint in response to bone-conduction stimulation Paper No II will show that apparently the second of these alternatives is the correct one

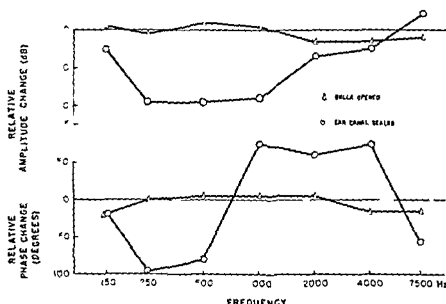



Fig 5 Amplitude changes and phase shifts for seven signal frequencies in reference to the normal ear resulting from the two procedures indicated (average of six animals)

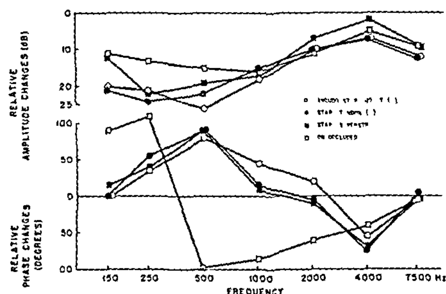



Fig 6 Same as Fig 5 for four additional procedures as indicated Results were obtained from the same group of animals shown in Fig 5

### 3 Amputation of the Middle Ear

The stepwise amputation of the middle ear (Fig 6) produced considerable amplitude losses mainly in the low frequency range The point of [illegible] l ss varied between 250 Hz and 1000 Hz The difference in re- [illegible] n the three situations were rather minor and, for the small n [illegible] sted could not be clearly separated from one another

However, the magnitude of the observed changes as such once more gave evidence for the ossicular inertial component of Barany (1938)

In the range of the largest amplitude losses, i e at the low to middle frequencies, there were positive phase changes of considerable magnitude (Fig 6, bottom) This latter finding is in line with the observations of Legouix & Tarab (1959) who saw positive phase shifts on impairment of middle-ear function

4 *Occlusion of the Oval Window*

The relative response curve following occlusion of the oval window (Fig 6) showed amplitude losses centered around 500 Hz and 1000 Hz It is noted, however, that these responses represent considerable gains, at least in the low to middle frequency range, with respect to the losses incurred in any of the three situations with the middle ear structures removed The phase-shift curve also set the effect of the oval-window occlusion clearly apart from those found in the other three situations

When compared to the results of oval window fixation obtained in the earlier study (Tonndorf & Tabor, 1962), the present losses turned out to be the least (Fig 7) This relative improvement coincided with the switch from zinc oxide to "Grip" cement which was said to provide a much superior bond to the underlying bone

In the present study, the middle-ear structures had been removed prior to the window occlusion In the earlier study, however, stapedial fixation had been carried out with the ossicular chain intact At that time an inverse relationship had been noted between the *quality* of the stapedial fixation and the *magnitude* of the resultant bone conduction losses In

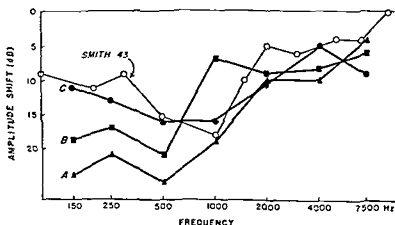



Fig 7 Bone conduction losses for seven frequencies due to different attempts to immobilize the stapes Curves A to C represent several stages through which stapedial fixation by means of dental cement, was gradually improved (cf text)

Also included in the graph are results of Smith (1943) who had immobilized the stapes by means of a thread slung around one stapedial crus

he earliest attempt had only succeeded in attaching a large lump of cement to the stapes, but had not achieved a fixation at all Yet it was the latter procedure which had produced the largest losses (Curve A of Fig 7) In a study conducted by Smith (1943), stapedial fixation had been achieved by means of a thread slung around one stapedial crus Although fixation was probably not very firm in this case, there was no mass added to the stapes Significantly, bone conduction losses in Smith's study had been approximately of the same order of magnitude as those obtained in the present experiments with the aid of "grip" cement (Curve C of Fig 7) Therefore, it appears that the losses observed in the earlier experiments of the present series (Curves A and B of Fig 7) were due to "stapedial loading" and not to the poorness of the fixation as such

The present results indicate that the relatively large losses observed by Brinkman *et al* (1965) might have been obtained under conditions of poor fixation

The explanation of the "stapedial loading effect" will be given in paper No II which is devoted to the loading effect of tympanic membrane to which stapedial loading appears to be related Suffice it to state at present that the explanation of the stapedial loading advanced earlier (Tonndorf & Tabor, 1962) cannot be maintained in view of the later findings

5 *Occlusion of the Round Window*

Occlusion of the round window with the middle ear intact had only small effects (Fig 8) This is in contrast to the earlier finding of Ranke *et al* (1952) However, it must be pointed out that the experimental technique of the latter study was quite different from that used here Relatively speaking the losses found in the present study were more pronounced in the high frequency region However, when the cochlear aqueduct was sealed

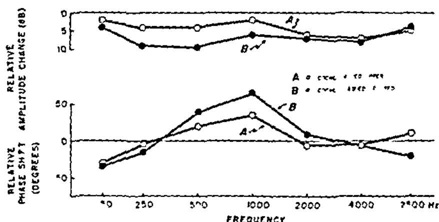



Fig 8 Amplitude changes and phase shifts produced by the occlusion of the round [illegible] even frequencies re normal ear In the first group of animals (same as [illegible] and Fig [illegible]) the cochlear aqueduct was left open In a second gr[illegible] [illegible] it had been sealed off

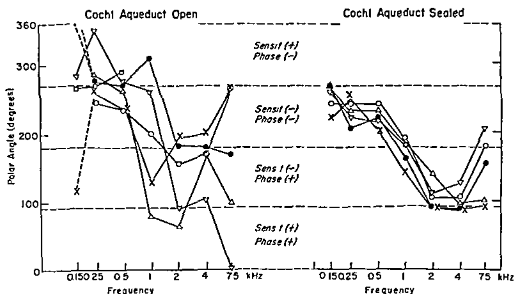



FIG 9 Polar angles for seven signal frequencies, produced by occlusion of the round window subsequent to (and in reference to) interruption of the incudo stapedial joint Left side cochlear aqueduct open right side cochlear aqueduct sealed (series of five animals each)

off simultaneously, the losses became slightly, but definitely larger, especially in the low frequency region (Fig 8, Curve B) The curve, thus acquired a positive slope The *leakage through the cochlear aqueduct* thus demonstrated had already been observed in the earlier study (Tonndorf & Tabor, 1962)

Groen & Hoagland (1958) had come to the same conclusion on the basis of a clinical observation The pre-operative bone-conduction audiogram of a patient with a confirmed otosclerotic occlusion of the round window had shown a negative slope, similar to that of Fig 8 Assuming the cochlear aqueduct to be acoustically patent, the authors advanced the following explanation The resistance through a narrow duct, being proportional to the velocity of movement through it, increases with frequency Hence the cochlear aqueduct ought to act as a low-pass filter The present findings are a direct confirmation of this earlier hypothesis of Groen & Hoagland

It is to be noted however, that the cochlear aqueduct was found to act as a leak *only* with respect to bone-conducted signals, but *not* to air-conducted signals (Tonndorf & Tabor, 1962)

Fig 9 shows another effect of sealing the cochlear aqueduct with respect to the round window occlusion The between-animal variability became

greatly reduced, indicating that the acoustic patency of the cochlear aqueduct must vary considerably between animals

In a more general form, the findings of Fig 8 had been predicted by Ranke (1958) He had suggested that *all* communications between the inner ear and the endocranial space be considered potential pathways of bone conduction He had coined the term "third cochlear window", by which he referred to the *sum total of* all *perineural and perivascular spaces* as well as the two aqueducts Ranke's notion was based upon earlier experimental findings of Jahn (1953). By contrast, Schneider (1959) had been unable to find any evidence for the "third window" effect in an experimental study of his own (An explanation for Schneider's negative findings will be given later, in connection with Fig 15 )

6 *Response Changes of the Isolated Inner Ear*

Fig 10 shows the effects of various procedures *in reference* to the bone conduction responses of the inner ear alone, i e after removal of the middle ear, including the stapedial superstructure (All data presented were obtained from animals in which the cochlear aqueduct had been sealed off Otherwise, occlusion of the round window or of both windows simultaneously would have been less effective )

The *occlusion of the oval window* (Fig 10) produced a *gain* in response amplitude in the low-frequency range (This fact had already been mentioned in connection with Fig 6 ) When observed earlier (Tonndorf & Tabor, 1962), this finding had been interpreted as resulting from closure of a *shunt* (or *leak*) on the vestibular side of the partition by way of the oval window

The *occlusion of the round window* (Fig 10) *decreased* the response amplitude, again mostly in the low-frequency range Although slightly smaller, the change was almost a mirror image of that due to the occlusion of the oval window Similarly, the phase shifts produced by the occlusion of either window (Fig 10, bottom) displayed opposite tendencies for most frequencies

Both of these findings support in principle the original concept of Herzog & Krainz (1926) concerning compressional bone conduction

The simultaneous occlusion of both windows (Fig 10) decreased the responses to levels slightly lower than those due to the occlusion of the round window alone It is noted that the difference between the effect of *the oval-window occlusion and that of both windows in* Fig 10 was somewhat larger than that seen in the earlier study under the same condition (Fig 8, loc cit ), i e presently 15 dB at the point of maximal separation at 250 Hz, as compared to only 9 dB previously, also at 250 Hz This increased difference was due to the lessening of the losses caused by the oval-window [illegible] (cf Fig 6) The phase shifts produced by the occlusion of both [illegible] [illegible]llowed those due to the occlusion of the oval window rather [illegible]

[illegible]

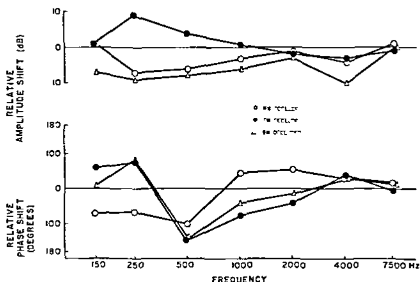



FIG 10 Amplitude changes and phase shifts for seven signal frequencies produced by occlusion of the two windows individually or together as indicated after (and in reference to) the following condition removal of the middle-ear structure, including the stapedial superstructure, cochlear aqueduct sealed (average of six animals)

These latter findings are *not* in agreement with the theory of Herzog & Krainz According to their concept, compliant windows, especially a compliant round window, are absolutely essential as releases of the incompressible cochlear fluids One might argue, of course, that most of the "*third window*" was *still open, since the present experiments were limited* to the closure of the cochlear aqueduct

Nevertheless, the losses with both windows *and* the cochlear aqueduct closed, as shown in Fig 10 were surprisingly small less than 10 dB for most frequencies It was doubtful, therefore, that the additional closure of the remaining "third window" would have really produced the profound loss predicted by the theory of Herzog & Krainz

These considerations prompted an examination of the fundamental question whether or not there might be bone conduction responses with *all* cochlear outlets closed Such a study, by necessity, could not be carried out in experimental animals It was conducted in cochlear models of the kind used by *one of the authors (J T)* on previous occasions In response to vibratory stimulation of such models, displacements of the cochlear partition occurred in the usual form of traveling waves (Tonndorf, 1962) Their origin was traced to periodic changes in the shape of the cochlear shell so-called *distortional vibrations* When, and only when, the partition divided the cochlear space asymmetrically so that one perilymphatic scala was larger than the other, the distortional vibrations caused the space occupied by either scala to change periodically and alternately As one scala grew

larger, the other one became smaller so that the partition had to yield in order to preserve the fluid volumes on either side of it. The displacement of the partition was thus found to be (a) independent of any window releases and (b) solely dependent upon the volume differences between the two perilymphatic spaces. Herzog & Krainz, as is recalled, thought *both* of these conditions necessary for the mechanisms of compressional bone conduction. Because of its mode of origin, the bone conduction component which was shown to function without cochlear windows will be referred to as the "pure compressional component".[1]

In order to show the interdependence of the (a) "pure compressional bone conduction", (b) the release through the oval window, and (c) that through the round window, Fig. 11 once more gives the data of Fig. 10, but replotted in reference to the condition with round *and* oval windows occluded and the cochlear aqueduct sealed. This experimental situation represents the "pure compressional component" as close as it can be realized in living animals. On first inspection, there seems to be one flaw in that *all* conditions including the opening of the oval window, are seen to lead to an improvement of bone conduction responses, although admittedly, the latter condition had by far the least effect.

The explanation lies in the following: included in Fig. 11 are the bone conduction responses after stapedectomy, a procedure which resulted in the highest gain at least in the lowest frequencies. Stapedectomy literally "takes the lid of the cochlear fluids", *i.e. it promotes the fluid-inertial component.* Fluid inertia can only function when there are releases on *both* sides of the cochlear partition. Complete blockage of all releases, even only on one side, must effectively eliminate the action of this component. It stands to reason that with the oval window opened, some inertial movement is activated due to *the incomplete elimination of the "third window"*, hence the slight improvement of responses upon opening of the oval window in Fig. 11.

Ranke (1958) expressed the opinion that the magnitude of the fluid inertial component should depend upon the volume difference between the two perilymphatic scalae, since the moment of inertia of the fluid column was determined by this difference. Following this suggestion, attempts were made in the present experiments to affect the bone conduction responses after stapedectomy by increasing or lessening the fluid level in the oval window. However, changes in level of up to 1.5 mm had no noticeable effects in this respect.

The *phase shifts* due to the opening of the round and oval windows respectively (Fig. 11 bottom) may be more revealing. When the round window was opened there were hardly any phase shifts (in contrast to the relatively large amplitude changes) indicating a *synergistic* action of the "pure compressional component" and the round window release. By

p. 8 on the subject of proper terminology

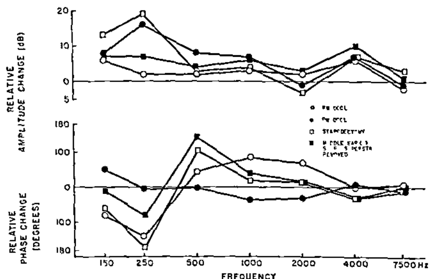



Fig 11 Data of Fig 10 replotted in reference to the condition with both windows closed simultaneously Results obtained after stapedectomy have been added (average of six animals)

contrast, phases were almost inverted when the release was shifted from the round window to the oval one This was a clear indication that the displacement pattern of the cochlear partition had changed its action in a like manner

Taken as a whole, the phase and amplitude data of Fig 11 suggest that the "pure compressional component" and the round-window release reinforce each other, whereas the oval window release and the "pure compressional component" oppose each other With the exception of the fact that these three are now recognized as three separate components, these results are in good agreement with the original postulates of Herzog & Krainz (1926) With respect to the role of the fluid inertial components, Fig 11 does not lend itself to such an obvious interpretation

*Compressional bone conduction*.—Mentioned earlier, in connection with Fig 8, was the fact that occlusion of the cochlear aqueduct, simultaneously with that of the round window, had an effect *only* upon *bone-conduction* responses but *not* upon *air-conduction* responses This finding has an important bearing upon the occurrence of compressional bone conduction Time and again, various authors, for example Wever & Lawrence (1954), have expressed their doubts that compressional bone conduction could be of real importance in the range of audio-frequencies, in view of the smallness of the cochlear spaces The present results indicate that with respect to the transmission of air borne signals, the cochlear aqueduct is not pat[illegible] In that case, the cochlear capsule must be considered a rigid shell How[illegible] in response to vibratory stimulation, the aqueduct, apparently, d[illegible]

come patent *Distortional vibrations* must occur altering its lumen periodically This, of course is the essence of compressional bone conduction and it is logical to assume that what had happened to the smaller aqueduct should also happen to the larger cochlear spaces

## E Analysis of Bone Conduction

The present results have permitted identification of seven different components all of which contribute in some measure to the total bone conduction response Most of them had been postulated by previous writers (In paper No X, the existence of an eighth component which originates in the external canal will be shown) The following components had been identified

(1) Middle ear ossicular inertia,
(2) Middle ear cavity compliance
(3) Pure compressional effect
(4) Oval window release
(5) Round window release
(6) Inner-ear fluid inertia
(7) Cochlear aqueduct effect

Some of these components may be further subdivided For example the small differences between the various steps of middle ear amputation as well as the *differences between the fixation of the stapes and that of the tympanic membrane had indicated that the middle ear inertial component could be broken down into several sub-components Furthermore* data had been collected to subdivide the middle ear cavity compliance into two sub-components its effect upon (a) the tympanic membrane and (b) upon the round window membrane This latter subdivision will be discussed later (cf Fig 16) At present the analysis will be limited to the seven components listed

Although simple inspection of the amplitude data such as those of Figs 5 or 10 and or phase data especially those of Fig 11 had given evidence for the action of the components listed such data are not always sufficient for purposes of a *quantitative* assessment It is apparent for example that the responses obtained after removal of all external and *middle-ear* structures represent those of the inner ear However the response of the middle ear cannot be assessed in such a direct manner The example of Fig 11 should make it clear that the different components combine with one another in *varying phase relationships* Consequently *vectorial subtraction* in the manner indicated in Fig 12 is needed for the determination of a component which cannot be assessed by direct measurement

Specifically the quantitative isolation of any given component was executed in the following manner First two experimental situations were [illegible]ch were identical save for the factor to be isolated The vectorial

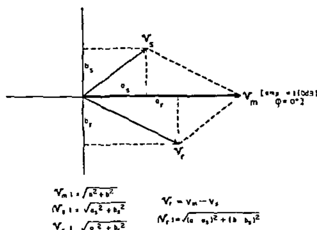



FIG 12 Vectorial subtraction For the sake of convenience the vector minuend ($V_m$) has been adjusted to an amplitude of one and a phase angle of zero Plotting the *linear* amplitude ratio of the vector subtrahend to the minuend ($V_s/V_m$) from the origin at its proper relative phase angle permits evaluation of the vector remainder ($V_r$) according to simple geometric rules If preferred i e for repeated operations or multiple subtraction the results can be *calculated* by the equations given below the graph

differences between the two sets of data, formed separately for each frequency, gave the desired results (In one instance, this calculation involved more than one step ) Since the results depend entirely upon the definitions employed, these are given in full in Table 2 For the sake of brevity, conventional symbols are used Vectors are written in **bold-face** type The symbols $\mathbf{V}_m$ $\mathbf{V}_s$ and $\mathbf{V}_r$, stand for vector minued, subtrahend and remainder respectively Abbreviations, such as *OW*, *BW*, *stap crura* (−) stand for experimental situations such as "closure of the oval window", of "both windows simultaneously", or "removal of the middle ear (including the stapedial superstructure)". For example, the notation $\mathbf{V}_r$ = *normal ear* − *OW* reads "the difference between vectors representing the normal ear and the situation after occlusion of the oval window" For proper evaluation of each component careful attention must be paid to the references as they appear in Table 2 After all components had been obtained numerically *re* normal ear, components #1 through #6 were once more added vectorially The idea was that if these six components really represented *all* factors contributing to the total bone conduction responses, their sum total should be approximately equal to the latter with due allowance for experimental errors (The reason component #7 was excluded from this summation was that it was contained, by definition, in #3) As Table 3 indicates, the deviations were reasonably small, the average deviation for all seven test frequencies being 9° of phase and −1 0 dB of amplitude Consequently, the definitions of Table 2 do not appear to contain any major errors — The above mentioned eighth component, which originates in the external canal, is presently contained in #3 the ossicular inertia Since it had not been identified from the experi-

TABLE 2. *Definitions*

| Component to be derived | Vector | Test result employed | Remarks |
|---|---|---|---|
| (1) Middle ear inertia | $V_m$ | Normal ear, bulla open | Contains all components except *middle-ear cavity compliance* |
| | $V_s$ | Stapedectomy (*re* normal ear) bulla open | Contains all inner ear components |
| | $V_r$ | *Difference* middle-ear inertia (*re* normal ear, bulla open) | *Note* Change of reference required |
| (2) Compliance effect of middle ear cavity | $V_m$ | Normal ear, bulla closed | Contains all components |
| | $V_s$ | Normal ear, bulla open | Contains all components except middle ear cavity compliance |
| | $V_r$ | *Difference* middle ear cavity compliance (*re* normal ear, bulla closed) | |
| (3) Inner ear compression | V | RW closed bulla closed 3rd window open (*re* normal ear) | No computation required |
| (4) Oval window release | $V_m$ | RW closed *re* stap crura (−) | Contains compressional effect & OW release |
| | $V_s$ | RW closed *re* stap crura (−) | Contains compressional effect only |
| | $V_r$ | *Difference* OW release {*re* stap crura (−)} | *Note* Change in reference required |
| (5) Round window release | $V_m$ | OW closed, *re* stap crura (−) | Contains compressional effect & RW release |
| | $V_s$ | RW closed *re* stap crura (−) | Contains compressional effect only |
| | $V_r$ | *Difference* RW release {*re* stap crura (−)} | *Note* Change in reference required |
| | | *Step 1* | |
| (6) Fluid inertia | $V_m$ | *Stap crura (−)* | *Contains all inner ear components* including fluid inertia & OW release |
| | $V_s$ | OW closed *re* stap crura (−) | Contains all inner ear components except fluid inertia & OW release |
| | $V_r$ | *Difference* interim result | Combination of fluid inertia & OW release |
| | | *Step 2* | |
| | $V_m$ | $-V_r$ | Combination of fluid inertia & OW release |
| | $V_s$ | $-V_r$ of #4 | OW release {*re* stap crura (−)} |
| | $V_r$ | *Difference* fluid inertia {*re* stap crura (−)} | *Note* Change in reference required |
| (7) Cochlear aqueduct release | $V_m$ | RW closed cochl aqueduct open (*re* normal ear) | *All components including aqueduct effect but excluding RW release & fluid inertia* |
| | $V_s$ | RW closed cochl aqueduct closed (*re* normal ear) | All components except RW release fluid inertia & cochlear aqueduct effect |
| | $V_r$ | *Difference* cochl aqueduct effect (*re* normal ear) | |

TABLE 3 *Re-Synthesis of the Total BC Response*

Deviations of calculated results (vectorial summation of six calculated components) from the total response

| Hz | 150 | 250 | 500 | 1000 | 2000 | 4000 | 7500 | Av |
|---|---|---|---|---|---|---|---|---|
| Ampl (dB) | − 1 | − 4 | −1 3 | − 1 | +3 6 | + 2 5 | − 3 2 | −1 0 |
| Phase | +54° | +16° | +4° | −29° | +1° | +13° | +16° | +9° |

mental data, it could not be isolated at this time However, this fact could not be detected from the calculations shown in Table 3 (For re-identification of component #3 after isolation of the component due to the external canal, cf paper No V )

Fig 13 shows the *calculated results* for the first six components listed above, both in terms of amplitude and phase They are presented separately as middle ear (Fig 13 *a*) and inner-ear (Fig 13 *b*) components respectively (The seventh, the cochlear aqueduct effect, will be dealt with separately later, cf Fig 15 ) It must be noted that all present results are given relative to a total bone conduction curve which, owing to the present lack of further information, was assumed to be flat, as assumption which is certainly not correct Consequently, presentation of components must be limited at this time to their *relative* contribution to the total bone conduction response However, no statements can be made at present with regard to the resonances and/or anti-resonances of the various underlying systems (For the actual response curves of these systems, cf paper No VI )

It is seen from Fig 13 *a* that, of the two middle-ear components isolated the *middle ear inertia* is by far the most important, especially around 500 Hz and 1000 Hz As a matter of fact, comparison with Fig 13 *b* indicates that, in the cat, this is the strongest of all components, except above 3000 Hz where the compressional component becomes gradually dominant The *middle ear compliance* effect is of lesser significance, except at the two extreme ends of the frequency range tested Individually, the contribution of the inner ear components to the total bone conduction response is not very large (Fig 13 *b*) The *round window release* and the *compressional component* stand out slightly from the two others The *oval window release* has very little importance in the low- and middle-frequency ranges Significantly, this is the same frequency region in which the ossicular-inertial component was found to be very strong (Fig 13 *a*) It appears therefore that with the ear intact the action of the latter minimizes the leak through the oval window The *compressional component* except at frequencies below 500 Hz, is a function gradually rising with frequency as was predicted by various previous writers, most recently by Kirikae (1959) (The absolute response curve, which will be given in paper No VI, actually fits this concept even better ) At frequencies approximately above 3000 Hz, the

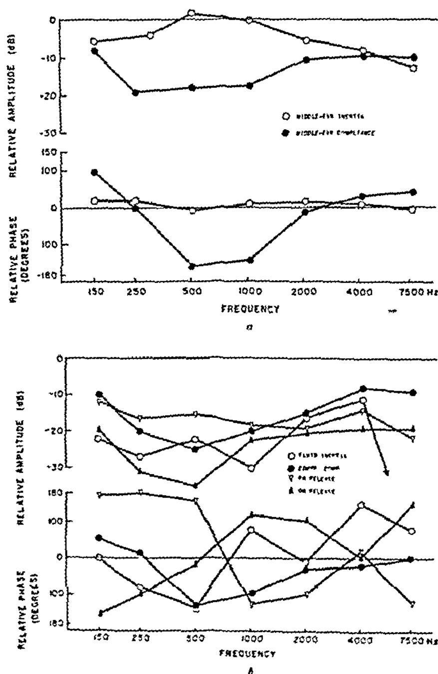



Fig. 13 Six bone-conduction components, calculated from the experimental results: amplitude and phase data for seven signal *frequencies*. *For the sake of better illustration* the graph has been divided into sections, *a* and *b*.

compressional component becomes the dominating one as already mentioned. The *fluid inertial* component contributes significantly only at frequencies around 2000 Hz and 4000 Hz and, to a lesser degree, at the lower end of the frequency scale.

Fig. 14 supports the *conclusion reached above on* the basis of the data

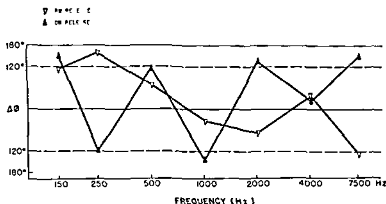



Fig 14 Phase differences for seven signal frequencies, of the two window releases, in reference to the compressional component of Fig 13 *b*
The limiting phase angle at which the sum of two vectors is made neither bigger nor smaller than either of them can be stated unequivocally only when both of them are equal in magnitude In that case the angle is 120° If they are unequal in magnitude, that of the larger one is preserved for phase angles smaller than 120°, but larger than 90° In the present figure the extreme value of 120° is taken to indicate the borderline between re inforcement and cancellation

of Fig 11, concerning the relationship between the compressional component and the two window releases This figure shows the phase relationships between each of the two window releases and the compressional component For the round window, the phase difference is smaller than 120° for most frequencies, indicating once more that the round window release reinforces the compressional component By contrast, for the oval-window release, the phase difference is larger than 120° for most frequencies Hence, the oval-window release *opposes* the compressional component

The *release through the cochlear aqueduct* is shown in Fig 15 The compressional component of Fig 13 *b* has been added once more for the sake of comparison Around its point of maximal contribution, at 1000 Hz, the effect of the aqueduct is, perhaps surprisingly, large At most other frequencies it is rather insignificant, although clearly demonstrable This finding demonstrates the principal soundness of Ranke's concept (1958) of the "third cochlear window" For obvious reasons, the total effect of the third window" could not be assessed in animal experiments, a fact which also precluded the exact assessment of the "pure compressional component" (In Fig 13 *b*, the latter was therefore given with the "third window" open ) The facts that (a) the oval and the round window *releases* oppose each other with respect to the compressional component (Figs 13 and 14), and that (b) the vestibular and cochlear aqueducts are also located opposite each other across the cochlear partition, i e the basilar membrane, makes it quite probable that a similar opposite rela-

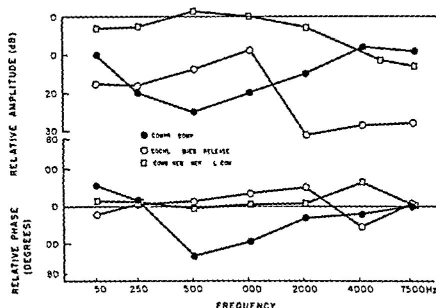



Fig 15 The pressure release through the cochlear aqueduct and the combined effect of the two inertial components (middle ear ossicular and inner ear fluid) amplitude and phase data for seven signal frequencies The compressional component of Fig 13 b is given once more for the sake of comparison

tionship exists between the two aqueducts If the effect of the vestibular aqueduct would be similar in magnitude to that of the cochlear aqueduct a reasonable assumption then the pure compressional component in the sense of the model finding (Tonndorf 1962) should be fairly close to the compressional component shown in Fig 13 *b*

Schneider (1959) as already mentioned was unable to find any experimental evidence for the third window effect However it must be pointed out that Schneider had employed but one frequency in his experiments namely 1000 Hz Fig 15 shows that the effect (at least for the cochlear aqueduct) happens to be rather small in that particular frequency range Moreover if the present concept is correct, i e that the effects of the two aqueducts oppose each other the total effect Schneider was searching for may be quite small

Also given in Fig 15 is the vectorial *combination of the two inertial components* that of the middle ear ossicles and that of the inner ear fluids This combination is most prominent in the middle frequency region gradually becoming less as frequency goes higher

Fig 16 shows the separation of the *middle ear cavity compliance* into its two sub-components calculated from the present data These sub-components are (a) the relative effect upon the tympanic membrane and (b) that upon the round window membrane Similar to the results of Groen 1962) who had calculated these two sub-components for the case of man the effect upon the round window due to its smaller surface area turned out to be the lesser of the two by an approximate average of 10 dB Other

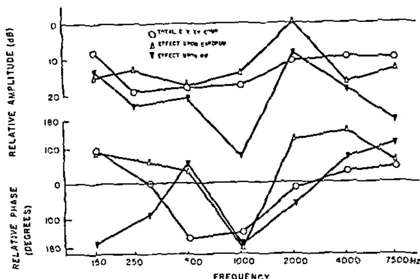



Fig 16 The middle ear cavity compliance and its two sub-components (a) the effect upon the tympanic membrane, and (b) the effect upon the round window membrane amplitude and phase data for seven signal frequencies

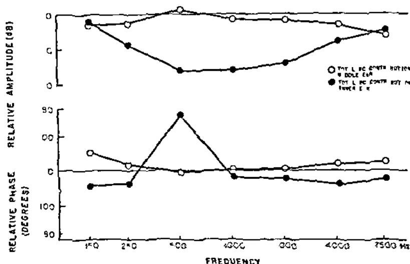



Fig 17 The bone conduction contribution of the middle ear and that of the inner ear amplitude and phase data for seven signal frequencies The middle-ear contribution represents the vectorial sum of the ossicular inertial component and the cavity compliance The inner ear contribution has been *calculated* as the vectorial difference between the total bone-conduction response and the middle-ear contribution

wise, the curves representing the two sub-components are rather similar to each other It is noted, however, that the combined effect lacks the rather sharp maximum at 2000 Hz of both sub-effects Once more, this is evidence for the fact that the combined effect of the two components cannot be predicted from mere inspection of their individual amplitude

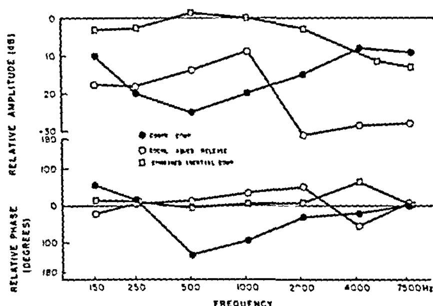



Fig 15 The pressure release through the cochlear aqueduct and the combined effect of the two inertial components (middle-ear ossicular and inner ear fluid), amplitude and phase data for seven signal frequencies The compressional component of Fig 13 *b* is given once more for the sake of comparison

tionship exists between the two aqueducts If the effect of the vestibular aqueduct would be similar in magnitude to that of the cochlear aqueduct, a reasonable assumption, then the "pure compressional component" in the sense of the model finding (Tonndorf, 1962), should be fairly close to the compressional component shown in Fig 13 *b*

Schneider (1959), as already mentioned, was unable to find any experimental evidence for the "third-window effect" However, it must be pointed out that Schneider had employed but one frequency in his experiments, namely 6000 Hz Fig 15 shows that the effect (at least for the cochlear aqueduct) happens to be rather small in that particular frequency range Moreover, if the present concept is correct, i e that the effects of the two aqueducts oppose each other, the total effect Schneider was searching for may be quite small

Also given in Fig 15 is the vectorial *combination of the two inertial components* that of the middle-ear ossicles and that of the inner-ear fluids This combination is most prominent in the middle-frequency region, gradually becoming less as frequency goes higher

Fig 16 shows the separation of the *middle-ear cavity compliance* into its two sub components, calculated from the present data These sub components are (a) the relative effect upon the tympanic membrane and (b) that upon the round-window membrane Similar to the results of Groen (1962) who had calculated these two sub-components for the case of man, the effect upon the round window, due to its smaller surface area, turned out to be the lesser of the two by an approximate average of 10 dB Other-

the release through the 'third window' Of the latter only a part, that occurring through the cochlear aqueduct has been verified experimentally [The later identification (cf paper No X) of the component due to the external canal did not affect the validity of the above classification In the present data this component was said to be contained in the middle ear inertial component but when properly isolated it turned out to be an important component in its own right ]

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# II LOADING OF THE TYMPANIC MEMBRANE ITS EFFECT UPON BONE CONDUCTION IN EXPERIMENTAL ANIMALS

JUERGEN TONNDORF AND ARNDT J DUVALL III

## Summary

Experiments on tympanic membrane loading have been conducted systematically in cats Some additional data have been collected in three other species dog rat and guinea pig In general the results were similar to those obtained in man an increase in bone conduction responses at low frequencies accompanied by a decrease at high frequencies both of these changes being in some proportion to the applied load

A correlation of these findings with the results of a quantitative analysis of bone conduction responses into its various components indicated that tympanic membrane loading may have the following effects It alters the moment of inertia of the ossicular system of the middle ear by (a) making its effective mass heavier and (b) gradually moving its center of gravity which normally appears to lie slightly above the rotational axis toward the tip of the *manubrium mallei* The restraint of this system is given by (a) its own suspension (b) the inertia of the inner ear fluids (c) the air enclosed in the middle ear and probably also (d) by the air column contained in the external canal

Loading of the tympanic membrane shifts the resonant curve of the combined system toward the left thereby lowering responses to all frequencies above the newly established resonant point Because of the concomitant increase in the effective driving force (due to the increase in the moment of inertia of the ossicular system) the response to all frequencies below the newly established resonant point is improved

## A Introduction

R Barany (1910) is usually credited with having been the first to observe that small weights attached to the tympanic membrane improve hearing by bone conduction This finding was confirmed in a number of later studies (E Barany 1938 Rytzner 1954 Kirikae 1959 Allen & Fernandez, 1960 Abu Jaudeh 1964 Brinkman *et al* 1965) Actually all of the more recent studies with the exception of Brinkman *et al* indicated

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that the improvement is limited to the low frequency region, while the response at higher frequencies is somewhat reduced. Because of its implication for the Theory of Bone Conduction, tympanic membrane loading was included as an experimental variable in a series of bone conduction studies conducted in this laboratory.

The response of the ear to bone conducted sound is known to result from the combined action of a number of bone conduction components. In an earlier study (cf. paper No. I) seven different components were determined quantitatively in the cat. Evidence for the existence of an eighth component will be presented in paper No. X.

E. Bárány (1938) concluded from his own experiments that the loading of the tympanic membrane affects both the *ossicular inertial component of the middle ear* and the *fluid inertial component of the inner ear*.

Briefly, the action of the ossicular inertial component, according to Bárány's account, is as follows. Within the range of audio frequencies the malleus and incus move *in phase*, i.e. the incudo-mallar joint is essentially immobile. (The latter fact was recently confirmed by Møller 1963.) The two ossicles have but one degree of freedom which facilitates a rotational movement around a common axis running from the anterior mallar ligament to the incudal ligament posteriorly. The common center of gravity lies slightly below the rotational axis. Consequently, when the skull is subjected to vibratory stimulation, the ossicles react essentially like a pendulum which is vibrated at its support. Since the structural properties of the pendulum (the ossicles) are quite different from those of the support (the skull) and the coupling is small, these two structures vibrate with different amplitudes and phases. The resultant relative movement manifests itself in a displacement of the stapes footplate in reference to the oval *window. Ordinarily, according to Bárány, the efficiency of the ossicular* system is not very high in this respect. The moment of inertia in response to translatory motion is quite small, since the distance from the center of gravity of the two ossicles to their axis of rotation is in the order to $3 \cdot 10^{-2}$ cm. Weights attached to the center of the tympanic membrane, i.e. to the handle of the malleus, must increase both the effective mass and its leverage and thus, by augmenting the moment of inertia, improve the response of the system.

Kirikae (1960) reported some results of his own which have bearings upon the problem under consideration. In human cadavers he found the axis of rotation of malleus and incus to run from the *lowermost* portion of the anterior mallar ligament to that of the incudal ligament. He thus placed the center of gravity of the two ossicles, which he also determined, slightly *above* the axis of rotation. According to a drawing of his (Fig. 83, loc. cit.) the distance is in the order of $1 \cdot 10^{-3}$ cm in the case of man. Kirikae argued further that the stapedial weight might be neglected because of its relatively small value (in man 2.3 mg *vs.* 50 mg of the combined malleus and incus). However, he did take the weight of the eardrum into considera-

tion, the addition of which, in his opinion, would bring the center of gravity somewhat closer to the rotational axis than is indicated in the above figure

Barany (1938), as already mentioned, was of the opinion that drum membrane loading would also affect the fluid inertial component of the inner ear (The existence of this component was confirmed in the study reported in paper No I) Kirikae (1959) explained the high-frequency loss observed upon loading the tympanic membrane as being due to the fluid inertia

Barany's concept of tympanic membrane loading was challenged by Allen & Fernandez (1960) who attempted to show that the *compressional* (inner ear) mode was the only one of real importance with respect to the overall bone conduction response However, their criticism was apparently based upon a misunderstanding Barany had stated that, because of its inertial nature, the ossicular bone conduction response should vary as the square of the frequency of the driving signal In the opinion of Allen & Fernandez, this relation should lead to a proportional increase of the loading effect with frequency, an assumption they were unable to verify What they had failed to realize was that Barany's statement applies only to the acting force, but not to the response of the ossicular system The latter is modified by its own impedance as will be shown later

## B Procedure

The experimental procedure was the same as that in the earlier studies (Tonndorf & Tabor, 1962, paper No I) Therefore, a brief summary will suffice

The main experiments were carried out in cats The animals were anesthetized by sodium nembutal (22 mg/kg weight) after premedication with chlorpromazine (14 mg/kg weight) Because of the lowering effect chlorpromazine has upon body temperature, the animals were kept warm by means of a heating pad Cochlear microphonic response, including both amplitudes and phases, were measured under standardized conditions for seven test frequencies in octave or near-octave steps between 150 Hz and 7500 Hz before and after the application of the loads to the tympanic membrane Bone conduction signals were applied by means of a Western Electric bone-conduction vibrator (D 80904) The signal amplitude was monitored by an accelerometer which was rigidly attached to the skull in close proximity to the ear for the duration of the experiment This method had been found very reliable for the assessment of *relative* changes

The animal's head was held loosely in position by means of a headholder so that the test ear pointed upward The pinna was amputated in order to permit good visual control during the loading experiments Known volumes of mercury, measured in microliters (*l*), were placed at the umbo Mul-

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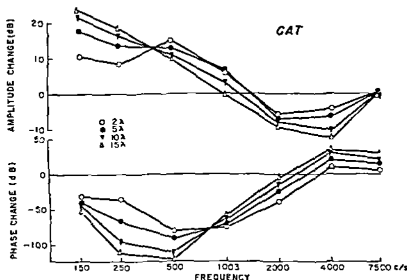



FIG 1 Amplitude and phase shifts resulting from tympanic membrane loading for seven frequencies (average of five animals) The loads are given in milliliters (λ) of mercury Thus 2 λ – 27 2 mg, 5 λ = 68 mg, 10 λ = 136 mg, 15 λ = 20[illegible] mg

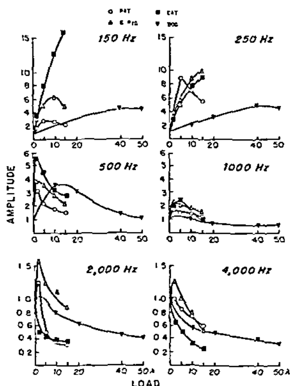



FIG 2 Response *vs* loads for six frequencies in the cat, dog rat, and guinea pig, as indicated (average of five animals in each species) Note that both ordinate and abscissa are given in *linear* values

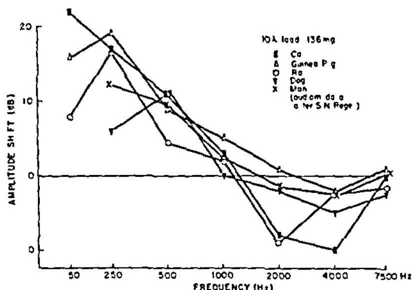



Fig. 3. Amplitude changes vs. frequency at a load of 10 $\lambda = 136$ mg in the four animals species. The results of audiometric measurements obtained by S. N. Reger (unpublished) under the same load are included for the sake of comparison.

and for relatively small loads amplitude increased linearly—or almost linearly—with the load. At the high frequencies amplitude appeared to depend approximately upon the inverse load. The curves for frequencies at which initial gains were followed by relative losses with further increases of the load (which included extreme load conditions at the low frequencies) were clearly combinations of these two functions.

There were some obvious species differences. In the present group the cat's ear appeared to be the most sensitive and the dog's ear the least sensitive with respect to tympanic membrane loading. Also, whereas in the cat the transition between initial gains and losses took place somewhere between 1000 Hz and 2000 Hz, this point of transition was found between 2000 Hz and 4000 Hz in the dog and beyond 4000 Hz in the rat and the guinea pig.

Fig. 3 gives a comparison among the four species. Frequency vs. amplitude changes are plotted at a given load level—viz. at 10 $\lambda = 136$ mg. Included in this figure is a curve which S. N. Reger obtained in normal hearing subjects by means of audiometric measurements for the same load (Reger unpublished). Generally speaking all curves are similar to one another indicating that tympanic membrane loading produces very similar effects in all of the four species tested as well as in man.

## D. Discussion

In a first attempt to put this data into some order the measurement of amplitude and phase obtained in the cat were combined in the form of vectors. Inspection of Fig. 4 immediately suggests simple descriptions of

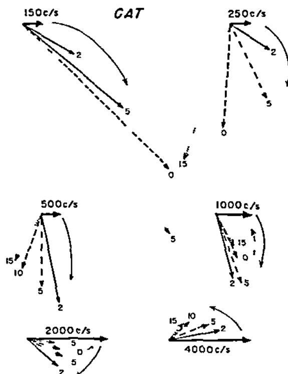



FIG. 4. Amplitude and phase shift data (cat) combined in the form of vectors. Note that the vector magnitudes are once more given in *linear* values. The reference vectors (zero load) have arbitrary lengths to suit the drawing.

the two lowest and the highest frequencies, i.e. of 150 Hz, 250 Hz, and 400 Hz respectively. (Note once more that the vector magnitudes are given in linear values.) The locus curves formed by the termini of the vectors for these three frequencies are *spirals*. For the two lowest frequencies these spirals run in a negative direction (clockwise in the graph) and their radii increase with the angle of deflection. The spiral describing the locus curve for 4000 Hz runs in the positive direction but its radius decreases with angular deflection. All three are thus seen to be sections of positive running spirals and can be fitted to hyperbolic spirals of the general form

$$a = r(\vartheta_0 - \vartheta) \tag{1}$$

(in equation (1) $a$ is a constant, $r$ is the radius of a given locus along the spiral having an angular deflection of $\vartheta$ with respect to the zero load condition, the latter being defined by $\vartheta_0$.)

The spirals for the three intermediate frequencies are complex. That for 500 Hz runs in a negative direction and its radius increases at first toward a maximal value in the vicinity of the 2 g load vector; thereafter the radius decreases again.

With respect to the spirals representing 1000 Hz and 2000 Hz there

appear to be two possibilities. Either they run in a positive direction all the way, thus having an extremely large interval of angular deflection between the zero load and the 2*l* load conditions, or they reverse themselves, running in a negative direction at first to attain a positive direction later. The second alternative is probably the correct one, as will be shown presently, and is therefore indicated in the graph.

At this point Bárány's mathematical considerations must be reviewed in some detail. As it was briefly mentioned above, he argued that tympanic membrane loading should affect both inertial components, i.e. the ossicular as well as the fluid inertial ones. However, loading should alter the ossicular system only, i.e. (a) its mass at zero load ($m_0$) and (b) its force arm ($d_0$) (i.e. the distance from the center of gravity to the rotational axis of malleus and incus) by increments $\Delta m$ and $\Delta d$ respectively. In line with the leverage laws, the resistance arm ($l$), i.e. the distance from the stapes footplate to the rotational axis, is inversely related to the force arm. Thus the force ($I$) acting upon the ossicular system according to Bárány may be written as

$$I = x\omega^2(m_0+\Delta m)(d_0+\Delta d)(l^{-1}) \qquad (2)$$

where $x$ stands for the amplitude of the driving system and $\omega = 2\pi f$ ($f$ = frequency). Since $(d_0+d)l^{-1}$ is a dimensionless factor and $\omega$ = sec$^{-1}$, it is easily seen that this equation has the dimension $MLT^{-2}$, i.e. the general properties of an inertial force. Equation (2) is the basic equation, the application of which Allen & Fernández had questioned because it seemed according to this equation that the force (and its effect?) should increase in proportion to the square of frequency.

However, the response of a given system is not determined by the driving force alone but by the ratio of this force to the restraint acting upon the system, i.e. its impedance. The impedance then includes the fluid inertial component. In combining several of Bárány's original equations one may obtain the stapedial displacement ($\xi$)

$$\xi \sim \frac{x\omega(m_0+\Delta m)(d_0+\Delta d)l^{-1}}{\{R^2+[\omega(m_f+\theta l^{-2})+D\omega^{-1}]^2\}} \qquad (3)$$

It is noted that the mass factor in the denominator is made up of the inner ear fluid mass ($m_f$) and the moment of inertia of the ossicular system ($\theta l^{-2}$). (The fact that the impedance is given as that of a simple series circuit may well be an oversimplification.)

Equation (3) is written here with a proportionality sign rather than an equal sign as Bárány had it. When equation (2) is re-written in dimensional form it becomes

$$\frac{MLT^{-2}}{MT^{-1}} \cdot L(T^{-1}) = |\xi\omega| \qquad (4)$$

In other words, results must be divided by $2\pi f$, before actual displacement values $\xi$ are obtained Equation (4) shows quite clearly that the objection of Allen & Fernandez had really been based upon a misunderstanding

If Barany's reasoning is accepted i e tympanic membrane loading affects the inertial components of the middle and inner ears, simple consideration of equation (3) suggests the following two postulates

(1) An increase of the vibrating mass $(m_0 + \Delta m)$ and of the force arm $(d_0 + \Delta d)$ ought to *augment the moment of inertia of the ossicular system* However, the increase of the mass factor must lower the resonant point of the system, i e *shift its response curve toward the left*

It follows from this first postulate that the *response ought to increase for frequencies lower than the newly established resonant point, but decrease for frequencies above that point* Generally speaking, this is a good description of the results shown in Figs 2 and 3 It might also account for the observations at middle frequencies, i e that for small loads responses increased, but decreased as the loads became higher At first, these frequencies might be located to the left and later to the right of the resonant point

(2) The numerator of equation (3) varies both with the increases of $m$ and of $d$ or

$$F = f(m, d) \tag{5}$$

However, the force arm $(d_0 + \Delta d)$ approaches a limit $(d_{max})$, specifically when its length becomes equal to that of the handle of the malleus Thereafter,

$$F = f(m) \tag{6}$$

only

It follows from this second postulate that at low frequencies the curve, representing the response change per frequency which alters as a function of the force $F$, should consist of two separate segments, the first given by equation (5) and increasing more steeply than the second one given by equation (6) The point of discontinuity between these two segments would be represented by $d_{max}$ The data (Fig 2) at 150 Hz and 250 Hz, especially for the cat indicate that they may well fit the present assumption instead of the continuous function against which they had been plotted

Before the present results can be examined numerically in the light of the above criteria it must be pointed out that all presentations of the loading effect in Figs 1 through 4 were given in reference to a zero load condition which represented the *total* bone conduction response the *resultant of all of its components* However if Barany s concept is correct, or that only the inertial components of bone conduction are affected by drum membrane loading, shifts re the total bone conduction response do not represent the actual picture The difference between the *apparent* load

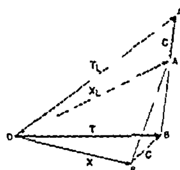



Fig. 5. Vectorial evaluation of the real and the apparent load effects (schematic). For explanation cf. text.

effect (*re* the total response) and the *real* load effect (*re* the component(s) actually affected) is illustrated by the schematic example of Fig. 5.

Suppose tympanic membrane loading affects one particular bone conduction component which in the zero load state and for a given frequency can be represented by the vector **X**. Since **X** will presumably differ from the vector representing the total bone conduction response **T**, all other components may be lumped together into a third vector **C** so that

$$\mathbf{X} + \mathbf{C} = \mathbf{T} \tag{7}$$

(Following conventional notation, vectors are given in **bold** face type.)

Under a given load condition **X** will be altered to $\mathbf{X_L}$. This real shift, however, will be obscured by the presence of vector **C** which by definition will be unchanged. Consequently it is the apparent shift

$$\mathbf{T_L} = \mathbf{X_L} + \mathbf{C} \tag{8}$$

which is actually recorded.

It may be noted from Fig. 5 that under two conditions, i.e. (1) that $\mathbf{X} > \mathbf{C}$ and (2) that $\mathbf{X_L} > \mathbf{X}$ (and, of course, $\mathbf{T_L} > \mathbf{T}$) the triangles $OAB$ and $OAB$ are very nearly similar triangles. That is to say in that case (the conditions actually depicted in Fig. 5) the apparent shift

$$\mathbf{T_L}(re\,\mathbf{T}) \simeq \mathbf{X_L}(re\,\mathbf{X}) \tag{9}$$

the real shift. A quantitative analysis of bone conduction components (paper No. I) indicated that for most frequencies the components of potential interest in this connection, especially the ossicular inertia, were so dominant as to fulfill the conditions of equations (8) and (9). This made it possible to use the original data in an effort to find which component or combinations of such might best fit the zero load condition under the conditions of the postulates made in connection with equation (3).

The relative components of bone conduction as derived in paper No. I were of course not suitable for this purpose. In a later paper (No. VI)

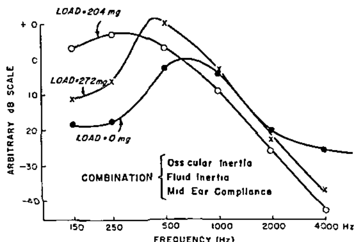



FIG 6 Amplitude data (cat) plotted as positive or negative changes in reference to the response curve of the system affected by the loading of the tympanic membrane The system which suited the experimental data best under the condition of equation (3) was the combination of (a) ossicular inertia, (b) middle ear compliance and (c) inner ear fluid inertia For the sake of clearer illustration only the two extreme load conditions are shown

the true response curves of each component will be given, and it is these which were utilized here It turned out that neither the ossicular inertia alone, nor the combination of both inertial components, as suggested by Barany, fitted the above criteria adequately when used as the zero load condition It was not until the middle-ear cavity compliance was included as a third factor that the curves of Fig 6 were obtained There is good reason for including the latter factor into the combined system, for it forms part of its restraint in the same manner as the fluid inertia component as Barany had assumed [In equation (3) it would be mainly represented by the compliance factor $D$ in the denominator ] Fig 6, for the sake of clearer presentation, is limited to the two extreme load conditions (2λ and 15λ) used in the actual experiments The two curves agree well with the criteria set up above The values at 7500 Hz were left out of this presentation, for they would have required large corrections in the sense of Fig 5 and equation (8) Even those at 4000 Hz may be in need of some corrections

The phase shift data (Fig 7) are in line with the interpretation of Fig 6 That is to say, they are in keeping with the assumption of a shift of the resonant point toward the left An impression that they might also indicate changes in damping of the affected system i e that damping might decrease with the load, could not be confirmed definitely. The question raised in connection with Fig 4 as to whether at 1000 Hz and 2000 Hz all phase changes occurred in the positive direction or they reversed themselves running negative first and attaining a positive direction later, can

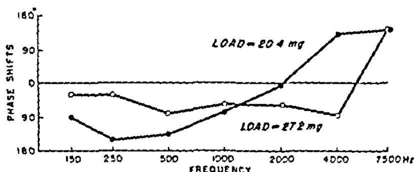



FIG 7 Phase shifts re the combined system of Fig 6 for the two extreme loads used

now be resolved At low loads, as long as the newly-established resonant point remains to the right of these frequencies, phase shifts must occur in the negative direction With higher loads, i e after the resonant point had moved to the left of these frequencies, phase shifts must be in a positive direction

In the other three species (dog, rat, and guinea pig), phase shift data had not been collected so that a complete analysis in the sense of Fig 6 was not feasible However, the loading effects were so similar in all species (Fig 4) that the underlying mechanism is quite probably the same in all of them And then again, some species-specific differences are worthy of comments

It was mentioned above, that the point of transition between initial gains at low frequencies and losses at high frequencies varied with the species, being lowest in the cat and highest in the rat and guinea pig Fig 6 indicates that in the unloaded state the resonant point of the combined system affected by loading the tympanic membrane is found between these two frequency ranges Paper No III will show that the differences among species with respect to the location of the point of transition is paralleled by the variation in the resonant point of the middle ear found in the same species, a point which lends further support to the analysis of the loading effect presented here

Fig 2 indicated that the dog was similar to the cat in that there was a shift of the resonance curve to the left in some proportion to the load as shown in Fig 6 In other words, the frequency for which the amplitude gain was maximal became continuously lower as the load was increased This did not hold true for the rat and the guinea pig In these two species, the maximal gain, once it reached the region of 250 Hz, remained confined to that region and did not extend farther down toward 150 Hz with further increases in load It appeared as if the system affected by the load possessed a low-frequency limitation in these two species (This statement, of course, does not imply that there is no such limit in cats and dogs, but only that if it exists it must be beyond the frequency and load range tested ) The explanation may lie in the following In the large

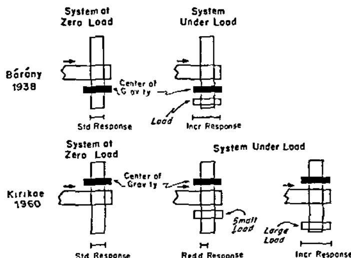



Fig 8 Schematic representation of the ossicular system in the unloaded and loaded states with its center of gravity placed according to Bárány (A) and according to Kirikae (B) The ossicular system is taken here as a pendulum the support of which is being vibrated in the direction shown by the arrow (This manner of presentation was originally used by Bárány ) The response to a given input is seen to vary with the alteration of the moment of inertia as indicated For further explanation cf text

animals (cats and dogs), the ball of mercury used for loading the tympanic membrane was always small compared to the diameter of the ear canal, even at the highest loads used It rested only upon the umbo and constituted nothing but a load acting directly upon the ossicular chain By contrast, in the smaller animals (rats and guinea pigs) the mercury balls, representing the larger loads of 10 γ and 15 γ, filled the entire ear canal Hence in these cases, the load acted partly upon the ossicular chain and partly upon the walls of the ear canal into which the ball of mercury was wedged This constituted a limit beyond which the effective load could not be increased (Actually, since the walls of the ear canal participate in the vibrations of the skull, so that its lumen changes periodically with the signal, as was found later (cf paper No V), the load effect with a drop of mercury wedged into the ear canal may even be more complicated than indicated in the foregoing )

It was mentioned above that Barany (1938) and Kirikae (1960) had come to opposite conclusions with regard to the location of the center of gravity of the middle ear ossicles Barany had placed it below and Kirikae above the axis of rotation If Barany were right, loading of the tympanic membrane should invariably increase responses in the low-frequency region If Kirikae were right, a small load should first decrease responses at these frequencies (However, there should be no concomitant increases at the high frequencies ) At higher loads, there should be the usual increases at low frequencies, and decreases at high frequencies These statements follow from consideration of the leverages involved (Fig 8) The

results of the present paper at first sight seemed to support Bárány's concept. However, the smallest load used (2.7 = 27.2 mg) was already very large with respect to the weights of the ossicles which are for the cat approximately 11 mg (malleus), 4.3 mg (incus) and 0.55 mg (stapes) according to our own measurements. Moreover, since the loads were applied at the umbo, i.e., at a considerable distance from the center of gravity (whether it be above the rotational axis or below it), the increase of the force arm and thus the change in the moment of inertia must have been considerable.

Data with lesser loads were available from an earlier study (Tonndorf & Tabor, 1963, paper No. I). They came from accidental observations concerning stapedial loading with incomplete fixation of the oval window (cf. Fig. 7, paper No. I). A lump of cement placed upon the stapes was found to weigh between 1 mg and 3 mg. Moreover, the moment of inertia produced by such a load was smaller than that due to a load placed upon the umbo by a factor of 1 : 2.5 (= −8 dB), the leverage ratio of the ossicular chain in cats (Wever & Lawrence, 1954). In cases of stapedial loading, there was a relative bone conduction loss in the low frequency range. This observation is hard to explain, except by assuming Kirikae's concept to be correct, i.e., that the center of gravity of the ossicular chain lies slightly *above* the rotational axis so that small loads decrease the response to bone conducted sounds.

An earlier attempt to explain the bone conduction losses observed upon stapedial loading (Tonndorf & Tabor, 1962) proved to be untenable in the light of the present findings.

As an alternative explanation for the phenomenon of stapedial loading, the possibility has been considered that a mass load upon the stapes might impair the transmission of the inertial middle-ear component. This explanation turned out to be unlikely. Cottle & Tonndorf (in press) were unable to produce significant transmission losses for *air-borne* signals by stapedial loading in cats. The maximal loss observed was a mere 9 dB at 4000 Hz for a weight of approximately 6 mg. Within the low frequencies, the region which is of interest here, the effect was even smaller.

## E. Comments

There are a few more points which may require some additional comments.

(1) It had been mentioned in paper No. I that unexpected phase variations occurred occasionally at 500 Hz, 1000 Hz, and to a lesser degree at 2000 Hz. This apparent phase instability can be explained in view of the present findings. It is noted in Fig. 4 that small loads produced large phase shifts at these frequencies. It is obviously the region around the resonant point of the unloaded system which is affected in this manner (Fig. 6). It is quite plausible that slight changes in the condition of the

tympanic membrane (e g the accumulation of a small amount of tissue fluids or blood in the ear canal) may at times have affected phase readings at the frequencies mentioned

(2) The curve fitting underlying Fig 6 did not take the compressional component of the inner ear into account One may wonder whether there should not be an effect upon the component labelled oval window pressure release especially since it is quite possible that the stapes was pressed into the oval window under the conditions of the present testing procedure First of all according to paper No I the oval window component is rather small and therefore its involvement might have escaped notice Secondly and more importantly Bekesy (1942) has shown that transmission of air conducted signals across the oval window is independent over a wide range of variations in intra labyrinthine pressure which also affects the position of the stapes It appears that the annular ligament can be stretched to quite some extent before transmission across the stapes is impaired

(3) The relatively low sensitivity of the dog s ear to tympanic membrane loading (cf Fig 2) may have two potential explanations (a) It is re called from equation (2) that the loads must actually be considered incre ments to the condition at zero load Thus it might be that in dogs the load increments are relatively small compared to $m_o$ and $d_o$ (b) It might also be that the contribution of the inertial component of the middle ear to the total bone conduction response is relatively minor in this species If the first alternative were correct large response amplitude should eventu ally be reached at low frequencies provided the loads were made high enough As Fig 2 indicated this assumption was obviously not correct Furthermore it was found later (cf paper No III) that in dogs the bone conduction losses produced by amputation of the middle ear were relatively slight Consequently the second alternative appears to be correct viz that in dogs the sensitivity to tympanic membrane loading is small because the contribution of the entire middle ear to the total bone conduction response is of lesser importance in this species than in the others tested

(4) It may be noted in Fig 2 that in the cat the load/response function at the two lowest frequencies is practically straight over a large range The explanation for this may lie in the following In the cat the handle of the malleus is known to be very long reaching well into the lower third of the *pars tensa* In line with equations (5) and (6) this must mean that the discontinuity in the load/response function at $d_{max}$ is not reached until relatively high load levels are attained

(5) The present experiments have indicated that tympanic loading affects three of the bone conduction components previously identified and quantita tively isolated However according to later experiments (paper No V) there is a fourth factor which although eliminated by the present pro- cedure ordinarily may also be affected Experiments concerning the occlu sion effect of the external ear canal had given evidence for the existence of a bone conduction component originating in the external ear canal

which, after its identification, was numerically isolated from previous data Without going into unnecessary details here, suffice it to say that from the standpoint of the present experiments, the air column of the external canal serves as a load upon the tympanic membrane in a manner similar to that due to the previously identified middle-ear cavity compliance Thus it appears to be safe to include the air column in the external ear canal into the list of components affected by tympanic membrane loading, although such effects had not been demonstrated in the current experiments

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# III. COMPARATIVE STUDIES IN BONE CONDUCTION IN CATS, DOGS, GUINEA PIGS, AND RATS

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AND RICHARD J. VOOTS

SUMMARY

The bone conduction losses resulting from the impairment of middle ear function (immobilization of the tympanic membrane and amputation of the middle ear) were compared in a series of four animal species: cat, dog, guinea pig and rat. The losses varied among the species both as to magnitude and the frequency value of the maximal loss. Correlation of these results with those obtained in experiments on loading of the tympanic membrane and with the analysis of bone conduction components, carried out in cats, suggested the following conclusions:

(1) Impairment (or elimination) of the ossicular inertial component is responsible for the losses observed. With respect to its contribution to the total bone conduction response, this component was most important in the guinea pig, next in the cat, in the rat, and least in the dog.

(2) The frequency value of the maximal bone conduction loss is indicative of the resonant frequency of the ossicular system, the value of which varied (from low to high) in the following order: cat, guinea pig, dog, and rat.

(3) The contribution to the total bone conduction response depends upon the moment of inertia for translational vibrations of the ossicular chain, whereby a large contribution is apparently associated with a low resonant point and *vice versa*.

(4) The resonant point of the middle ear when responding to air-conducted signals depends upon the properties of the ossicular inertial system as well as upon those of the compliance of the air within the middle ear cavities. By contrast, the resonant point of the middle ear, when responding to vibratory stimulation, is essentially determined by properties of the ossicular inertial system alone. Therefore, the two resonant points do not necessarily coincide in every species.

## A. INTRODUCTION

It had been noted in earlier bone conduction studies conducted in cats (Tonndorf & Tabor 1962, paper No. I), that the bone conduction losses

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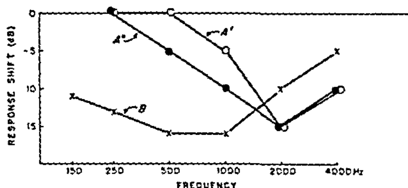



FIG 1 Bone conduction losses due to stapedial fixation in man and cat Curve A' individual clinical patient, the losses are plotted in reference to the status six months after stapedial mobilization Curve A" typical Carhart's notch Curve B amplitude losses in cats (cochlear microphonics) due to stapedial fixation made by means of dental cement (average of six animals)

due to amputation of the middle ear or due to stapedial fixation involved a *frequency range* which is considerably lower than that incurred in man under the same circumstances Initially, it had seemed that the *magnitude* of the bone conduction losses in the cat was also larger than that seen in man However, after the technique of stapedial fixation in cats had been perfected, this was no longer found to be true To state the problem differently Carhart's notch (Carhart, 1950, 1962), as the phenomenon is known clinically, occurs in a frequency range which is lower in cats than in man

Figs 1 and 2 show the apparent differences in a quantitative manner, i e the bone conduction losses incurred in the two species following stapedial fixation (Fig 1) and following amputation of the middle ear (Fig 2) The cat data represent the response shifts of cochlear microphonics due to the two conditions named The human data represent the results of audiometric measurements The loss due to stapedial fixation is given for one individual patient in reference to the status six months after successful stapedial mobilization with almost perfect restitution of hearing The typical Carhart's notch, the average of a large number of cases, is also included The loss due to amputation of the middle ear is illustrated by the bone conduction response shift, in reference to the pre-operative audiogram observed in a single patient on the sixth day after a radical mastoidectomy (cholesteatoma) at the time the surgical packing was first removed from the cavity Pre-operatively, the hearing had only been slightly reduced The post-operative audiogram was taken as early as possible in order to facilitate meaningful comparison of the human data with those obtained in acute animal experiments

Oddly enough a search of the literature failed to turn up any evidence ... tudies of hearing for bone conduction following radical

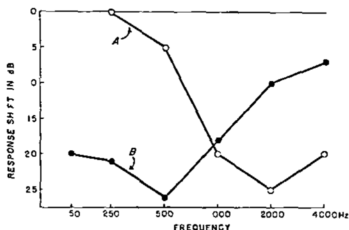



FIG 2 Bone conduction losses due to amputation of the middle ear in man and cat Curve A individual clinical patient the losses were measured on the sixth post operative day (radical mastoidectomy) and are plotted in reference to the pre operative audiogram Curve B amplitude losses in cats (cochlear microphonics) after middle-ear amputation (average of six animals)

mastoidectomy Only one other isolated case was found which Bekesy (1939) had once cited without giving any details as to how and under what circumstances the data had been obtained Nevertheless Bekesy s data were fairly similar to those presented here

Figs 1 and 2 indicate that in both instances the maximal loss has a frequency value in the vicinity of 2000 Hz in man whereas in cats it is closer to 500 Hz The magnitude of the loss is about the same in both species although in both species it is consistently larger after middle-ear amputation than after stapedial fixation

One may certainly argue that the differences between man and cat shown in Figs 1 and 2 could be due to the different techniques of assessment Not satisfied with such a simple explanation the present writers decided upon a systematic investigation of the problem at hand in four different animal species cat dog rat and guinea pig It was hoped that species specific differences if found might shed some light upon the contribution to bone conduction by the middle ear

The role of the middle ear in bone conduction was clarified mainly by the work of Barany (1938) Briefly when the skull is vibrated the middle ear ossicles being only loosely coupled to the skull will participate in this vibration due to their own moment of inertia with respect to translational motion This mode of response is therefore called *ossicular inertia* The existing phase and amplitude differences bring about a relative alternating displacement between the frame of the oval window (which is part of the skull) and the stapes footplate (which is part of the ossicular chain) and this in turn results in stimulation of the inner ear in essentially the

same manner as that following transmission of air-borne sound across the ossicular chain

Groen (1962), pointed out that this response of the ossicular chain is modified by the action of the air enclosed in the middle ear. The latter acts like a spring load upon the tympanic membrane, and to a lesser degree, upon the round-window membrane the *middle-ear compliance effect*

Lastly, a mobile stapes footplate constitutes a *leak* of the inner-ear compressional component the *oval window release*

In cats, the existence of all three of these factors was verified experimentally Subsequently, they were quantitatively isolated (paper No I) With respect to the overall bone conduction response, the relative contribution of the ossicular inertial component was found dominant up to approximately 1000 Hz, with a maximum around 500 Hz (cf Fig 13, loc cit ) The middle-ear compliance effect contributed considerably less and its curve did not form a clear-cut maximum although its contribution was generally stronger in the region of high frequencies than in that of the low ones The total contribution of the middle-ear (cf Fig 17, loc cit ), as might be suspected from Figs 1 and 2 of the present paper, dominated that of the inner ear except at the highest frequencies, the maximal contribution being once more at about 500 Hz The reason for the observation (Figs 1 and 2) that the loss following amputation of the middle ear was consistently more severe than that due to stapedial fixation may be explained by the fact that in the former case, the oval window release was acting as a leak, but in the latter case it was prevented from doing so With respect to the total response, the oval window release was of very little importance, its relative contribution being once more stronger in the high-frequency region than in the low-frequency region

## B Aim and Procedure

It was planned to study the effect of two kinds of experimental alterations of the middle ear upon bone conduction responses in the four species named (1) fixation of the tympanic membrane and (2) amputation of the middle ear The alterations resulting from a third effect, the loading of the tympanic membrane, have been reported separately in paper No II, since they concerned a set of questions of a somewhat different scope Briefly, it was found that the placement of small weights upon the tympanic membrane, i e in essence upon the tip of the *manubrium mallei*, affected the vibratory properties of the middle ear, in the sense of a *mass-load upon the ossicular system*

Fixation of the tympanic membrane, instead of the stapes, was chosen for the following reason even in cats, a reliable fixation of the stapes had not been easily accomplished In guinea pigs, in which the two windows are closely adjacent and especially in rats with their huge persistent stapedial artery, the prospect of working out a reliable technique of

stapedial fixation was deemed poor Moreover, it had been found in cats that the effects of immobilizing the tympanic membrane upon *bone conduction* responses were fairly similar to those caused by stapedial fixation That is to say, amplitude changes were not too different in the two cases However, phase shifts occurred in opposite direction indicating that, in contrast to the stapedial fixation, the immobilization of the tympanic membrane still allowed some mobility of the ossicular chain Two *alternative* explanations were thought to exist The mobility might be due to the fact that (a) not all of the tympanic membrane is tightly coupled to the ossicular chain, or (b) due to a yielding of the incudo stapedial joint, which ordinarily, i e in the transmission of air-borne sound, is known to be quite rigid (Møller, 1963)

The details of anesthesia, animal preparation, placement of the bone conduction vibrator, etc, were essentially the same as those of the earlier studies (papers No I and II) Responses were once more recorded in terms of cochlear microphonics at low levels (in the order of 1 μv) in order to avoid nonlinear effects In cats and guinea pigs, differential sealed-in electrodes in the basal turn were used, in dogs and rats single round-window electrodes were employed An indifferent electrode was placed into the neck muscles

The rat posed some special problems To begin with, the microphonic output *via* round window electrodes was found to be comparatively low Moreover, a headholder for proper placement of the head under the bone conduction vibrator was found to restrict the mobility of the head too much Therefore, a plasticine block was made with a suitable impression of the animal's head to assure proper lateral fixation This mold in turn was placed upon a foam rubber cushion to provide sufficient mobility in the plane of action of the bone vibrator Lastly, it was learned that the entire temporal bone of the rat is connected to the other skull bones by a rather loose fibrous union The same anatomical variation is known to exist in whales (Reysenbach de Haan, 1958) and in bats (Henson, 1961) With (a) the vibrator placed upon the parietal bone, (b) the temporal bone fixed to the parietal and occipital bones by means of "grip" dental cement (Caulk & Co) and (c) the head itself placed upon a resilient support, responses at the 1 μv level were obtained without exceeding the limits of linearity of the driving system (or of the microphonic input output function for that matter), even when the responses were reduced, for example after amputation of the middle ear

The large persistent stapedial artery of this species was already mentioned It runs close to the round window anteriorly and inferiorly, and must be handled with care Its venous mantle bleeds easily upon only slight provocation

Three to five animals of each species served to establish the procedure Six animals of each species were then run in succession and their results evaluated

## C Results

The results averaged for each species are presented in Figs 3 and 4 It is seen that after fixation of the tympanic membrane the magnitude of the maximal loss was slightly less and its frequency value slightly higher than after amputation of the middle ear This general statement is correct for all four species However under either condition neither the magnitude nor the frequency values at the maximal point of the induced loss were identical for the four species With respect to the severity of the losses animals may be classed in the following order from large to small guinea pig cat rat and dog (The reason why the guinea pig was placed first will be explained later) With respect to the frequency value of the maximal loss the rank order (from large to small) alters slightly cat guinea pig dog and rat

The present experiments were not designed for a complete assessment of the bone conduction components in all four species in the manner in which it had been done in the cat at an earlier occasion (paper No I) However the detailed information available for the cat permits one to ask some pertinent questions and to derive general conclusions from the present material

*Magnitude of the loss* —The question may be raised as to whether the magnitude of the observed losses might not be related to the relative contribution of the middle ear to the total bone conduction response Conceivably this contribution is not identical in all species In the cat in which the middle ear contribution to the bone conduction response was said to be dominant at low to middle frequencies (cf paper No I) middle ear amputation and immobilization of the tympanic membrane were seen to produce relatively large losses In the dog these losses were much smaller Moreover loading the tympanic membrane had produced much smaller effects in dogs than in cats and tympanic membrane loading had been shown to affect primarily the ossicular system (paper No II)

It might be mentioned in passing that the gain observed in dogs at 4000 Hz due to immobilization of the tympanic membrane (cf Fig 3) most likely has the following explanation Results of paper No I indicated that such an event takes place whenever the two components in question (middle and inner ear contributions in this case) are of the same order of magnitude but in or near phase opposition When acting together they partially cancel each other whereas after elimination of one component the other by itself produces a larger response

A comparison of the data obtained for cats (Figs 1 4) reveals that the losses following stapedial fixation were somewhat less than those after immobilization of the tympanic membrane and the latter in turn less than those following amputation of the middle ear The results of paper No I indicated that the larger losses were caused by a leakage of compressional bone conduction *via* the oval window when the stapes was left mobile It

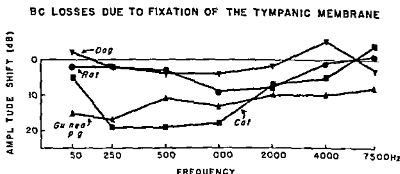



Fig 3 Bone conduction losses due to the fixation of the tympanic membrane in four animal species cat dog guinea pig and rat (average of six animals each)

appears then that in the cat, the immobilization of the tympanic membrane did not eliminate the oval window leak as efficiently as did stapedial fixation The same situation should prevail in the other species with the possible exception of the guinea pig i e the bone conduction losses due to stapedial fixation (which were not assessed in the present study) should be slightly less in magnitude than those produced by immobilization of the tympanic membrane

In the guinea pig the situation may be somewhat different A comparison of the magnitude of the losses shown in Figs 3 and 4 indicates that in this particular species the difference was least between the two conditions explored The guinea pig, as is well known does not possess a mobile incudo mallar joint The two bones are completely fused together It appears that it is this anatomical variation of the guinea pig s ear which made immobilization of the tympanic membrane more efficient with respect to eliminating oval window leaks than in the other species in which the incudo mallar joint is mobile Consequently it may be assumed (although this assumption has not been proven) that in the guinea pig the bone conduction losses due to immobilizing the tympanic membrane are ap

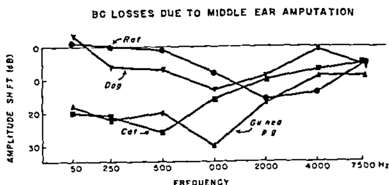



Fig 4 Bone conduction losses due to the amputation of the middle ear in four animal species cat dog guinea pig and rat (average of six animals each)

proximately equal to those due to stapedial fixation and it was for this reason that in the above list concerning the severity of the bone conduction losses produced by impairments of the middle ear function the guinea pig had been placed at the bottom These findings in the guinea pig support the second of the two notions expressed above, i e that the leak of compressional bone conduction following immobilization of the tympanic membrane in cats is due to a yielding of the incudo-malleolar joint rather than due to the incomplete coupling of the tympanic membrane to the ossicular chain

The present results have shown that the magnitude of the observed loss is indicative of the relative contribution of the middle ear to the total bone conduction response Hence the latter appeared to be largest in guinea pigs, and less in cats rats and dogs in that order

*The frequency value of the maximal bone conduction loss* —It is recalled from Figs 3 and 4 that the frequency value of the maximal bone conduction loss had varied with the species in the following order cat guinea pig dog and rat In the cat the two middle ear bone conduction components had been found to contribute maximally to different frequency regions i e the ossicular inertia at about 500 Hz and the middle-ear compliance at frequencies above 2000 Hz [In man, according to Groen's calculations (1962) the resonant point of the middle ear cavity appears to be at about 2500 Hz ] The question was therefore raised whether the species variations of Figs 3 and 4 with respect to the frequency value of the maximal loss might not be due to a difference in the relative importance of the two middle-ear components in making up the total middle-ear contribution

For each species the bone conduction losses due to middle ear amputation were compared with the bulla closed and opened i e with the compliance effect acting or eliminated It turned out that opening or closing the bulla did not affect the frequency value of the maximal bone conduction loss in an appreciable manner in any of the four species (How small the effect actually is may be taken from Fig 5 of paper No I ) Significantly, this was also true for the rat, the animal in which the maximal bone conduction loss had occurred at the highest frequency value i e at 2000 Hz These results indicate that the differences among species with respect to the frequency value of the maximal bone conduction loss as shown in Figs 3 and 4 cannot be due to a variation in the relative importance of the middle ear bone conduction components

The question of whether or not the presence or absence of the pinna might have an effect with regard to the present problem was next examined As Fig 5 shows for the example of the guinea pig there were small differences between the two situations but again the frequency value of the maximal bone conduction loss was not affected

The two findings that (a) the air enclosed in the middle ear and (b) the air column of the external canal have no decisive effect upon the bone conduction loss curve resulting from impairment of middle ear function

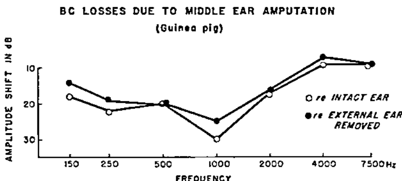



Fig 5 Bone conduction losses due to the amputation of the middle ear in the guinea pig (average of six animals) (a) with reference to the intact ear and (b) with reference to the condition after removal of the pinna and external canal

indicate that the important factor in this respect is really the response of the ossicular system In other words *it is essentially the missing ossicular inertial component which determines the frequency value of the point of the maximal bone conduction loss upon the amputation of the middle ear, immobilization of the tympanic membrane, or stapedial fixation*

*Carhart's notch* —Such an explanation of Carhart's notch runs somewhat counter to the accepted notion that the middle ear contribution to bone conduction is confined to low frequencies and becomes gradually smaller as frequency goes higher This concept is too simple It overlooks the fact that the ossicular chain, as any other vibrating system must have a resonant point and it is apparently the frequency of this resonant point which varies from species to species In the cat, the only species in which it was directly measured this resonance point was found at 600 Hz as will be shown later in paper No VI Interpretation of the present results (Figs 3 and 4) permit locating it approximately at 1000 Hz for guinea pigs and dogs and at 2000 Hz for rats By the same token (Figs 1 and 2) in man it should be in the vicinity of 2000 Hz

The reason that the resonance point may be as high as indicated here lies *in the fact that the moment of inertia* of the ossicular *system for the* translational motion is quite small as the calculations of Barany (1938), the anatomical measurements of Kirikae (1960), and the loading experiments of paper No II have indicated The center of gravity of the two major ossicles is quite close to their common rotational axis (probably slightly above it, according to Kirikae's findings which were confirmed by the results of paper No II) so that their effective mass and the resultant force are really small

The reason for the diversity among the four species tested probably lies in subtle anatomical differences No systematic studies were undertaken in this respect Nevertheless it is noted that the cat, for example, the animal with the lowest value of the resonant point possesses a very long

TABLE 1.

| | | | | |
|---|---|---|---|---|
| Freq value of max loss (from low to high) | Cat | Guinea pig | Dog | Rat |
| Magnit of BC loss (from large to small) | Guinea pig | Cat | Rat | Dog |

mandibular handle which reaches well into the lower third of the tympanic membrane According to Wever and Lawrence (1954), the leverage ratio of the ossicles is 1 2 5 in the cat, which contrasts sharply with the value of 1 1 3 found in man

It had been learned in paper No II that loading of the tympanic membrane increases the moment of inertia of the ossicular chain, thereby improving the magnitude of its vibratory response and simultaneously shifting its resonant point toward lower frequency values In most species, the resonant point of the ossicular system should be in the vicinity of the frequency value of the maximal bone conduction loss found upon impairment of middle ear function, a point which was eventually proven for the cat (paper No VI) It stands to reason, therefore, that in those species in which the above frequency value was low the relative contribution of the middle ear to the total bone conduction response should be high and *vice versa* It is seen from Table 1 that this is not an unreasonable assumption

*Middle ear impedance*—The notion that the bone conduction response of the middle ear in various species results from variations of the moment of inertia of the ossicular chain suggests that the same structural feature might cause a similar species specific variations with respect to the transmission characteristics of the middle ear to *air-borne* sound [The transmission properties of the ear have been studied quite extensively by Zwislocki (1957, 1962, 1963) and by Møller (1960, 1961 1963) ] It was decided to compare the middle ear contribution to bone conduction and the middle-ear impedance to air borne signals (the latter defined as acoustic impedance = dyne sec cm [5]) The comparison was limited to the location of the resonant point which, with respect to the bone conduction response, is approximately given by the points of maximal losses in Figs 3 and 4, and, with respect to the acoustic impedance, is represented by the point of minimal reactance

For assessment of the acoustic impedance, the bridge designed by Møller (1960) was utilized The experimental details are given in Appendix A Figs 6 to 9 give the reactance values for the four species, each figure representing data from several animals [The sudden aberrations from the smooth curves, according to Mundie (1960, 1962), are caused by momentary chang [illegible] impedan [illegible] are mainly due to alterations in the state of t [illegible] ear mu [illegible] The points of minimal reactance, indicative of r [illegible] were [illegible] tely as follows cat (bulla open)

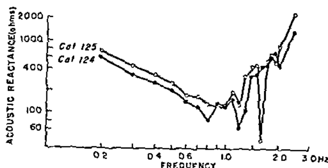



Fig 6 Acoustic reactances of the middle ear for various frequencies in two cats (bulla open)

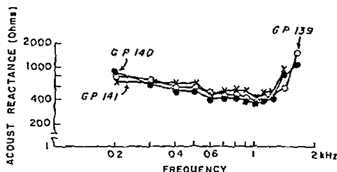



Fig 7 Acoustic reactances of the middle ear for various frequencies in three guinea pigs (bulla open).

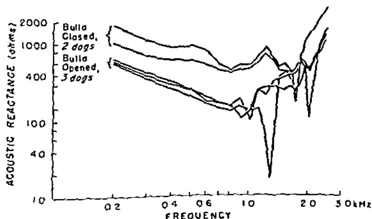



Fig 8 Acoustic reactances of the middle ear for various frequencies in two dogs (bulla closed) and three others (bulla open)

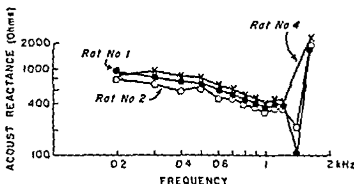



FIG 9 Acoustic reactances of the middle ear for various frequencies in three rats (bulla closed)

1000 Hz, guinea pig (bulla open)—1000 Hz, dog (bulla closed)—1600 Hz, (bulla open)—1000 Hz, rat (bulla closed)—1400 Hz.

Since the above data had been collected (1962), such measurements in experimental animals were reported by Møller (1963) for the cat and by Zwislocki (1963) for the guinea pig Figs 10 and 11 compare their data with those of the present experiments in terms of the total acoustic impedance

Møller's data (Fig 10) are in relative terms so that magnitudes cannot be compared However, he obtained data with the bulla open and closed Points of minimal impedance were at 1000 Hz and 2000 Hz respectively There was a second set of minimal points in the vicinity of 5500 Hz in both instances

Zwislocki (Fig 11), in cats, found a first minimum at about 2000 Hz and a second one at 7000 Hz In terms of magnitude, the values were somewhat higher than those of the present study The bulla was closed in his experiment He cited another set of data which is included here They were obtained by Eldredge by means of cochlear microphonic responses As Møller (1963) has recently shown, cochlear microphonic responses for a given sound pressure are proportional to the velocity of the cochlear window displacement Since

$$\frac{p}{v} = Z_{ch} \qquad (1)$$

($Z_{ch}$ = characteristic impedance dyne sec cm³), this means that the sound pressure necessary to produce a constant cochlear microphonic output, as in Eldredge's data (Fig 11), is proportional to the impedance Maximal sensitivity of the microphonic responses was approximately at 750 Hz with the bulla open and at 2000 Hz with the bulla closed Also, sensitivity in the first condition was somewhat better than in the second [Benson & Eldredge (1955) had earlier drawn attention to the difference in microphonic responses with the bulla open and closed ] Except for the fact that the curve for the present data turns up too steeply at its high frequency end, the two sets of data (A and B of Fig 11), are very similar to each other,

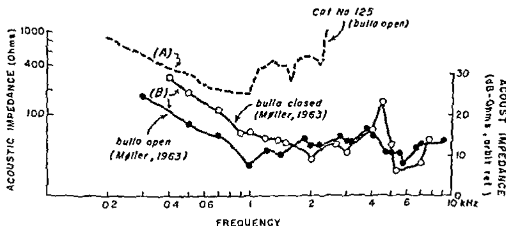



Fig 10 Acoustic impedances of the middle ear for various frequencies Curve A in one individual cat (own data) (bulla open), curves B according to measurements by Møller (1963), *with the bulla open and closed (relative impedance values)*

i e when the bulla is opened the curve shifts to the left and downwards The same is seen in Figs 8 (dog) and 10 (cat)

*Table* 2 is compiled from Figs 2, 4, 6, and 11 It compares the resonance points of the middle ear in the four species for air and bone conduction, with the bulla open and closed It is obvious that the bone conduction data correlated fairly well with those for air conduction with the bulla open, *but* not with those with the bulla closed The latter were consistently

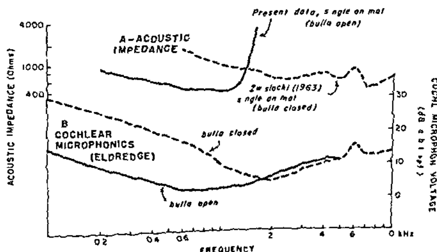



Fig 11 Acoustic impedances of the middle ear and cochlear microphonic response curves for various frequencies in guinea pigs Curve A own impedance data (bulla open) and data by Zwislocki (1963) (bulla closed) Curves B microphonic response curves with *bulla open and closed* data were obtained by D H Eldredge (cited after Zwislocki 1963

TABLE 2.

| | $f_0$ for air conduction | | $f_0$ for bone conduction |
|---|---|---|---|
| | Bulla closed (Hz) | Bulla open (Hz) | Bulla closed or bulla open (Hz) |
| Cat | 2000 | 1000 | 800 |
| Guinea pig | 2000 | 1000 | 1000 |
| Dog | 1700 | 1000 | 1000 |
| Rat | 1400 | ? | 2000 |

higher (In the rat, no data for air conduction with the bulla open are available)

It thus appears that the *transmission of air-borne signals through the middle ear is determined by properties of both the ossicular chain and of the middle-ear cavity compliance* (as Møller and Zwislocki had shown earlier), *whereas the response of the middle ear to vibratory (bone-conducted) signals depends mainly upon the properties of the ossicular system*

The lesser role of the middle-ear cavity compliance with respect to the bone conduction response of the middle ear had to be expected from the results of the analysis of bone-conduction components (cf Fig 13, paper No I)

Actually, it was not to be expected that the resonant properties of the middle ear should be the same for air and bone inputs Although the structural elements are the same in both cases, their arrangements differ Fig 12 attempts to illustrate this point with the aid of a simplified electric analogue of the kind Møller (1961) and Zwislocki (1962), have used in a more elaborate manner The R–C input ($Z_g - C_{dm}$) of the air-conduction analogue circuit represents the necessary step-up transformer (air/cochlear fluids) and, as such, is a low-pass filter The transformer-coupled input, representing inertial vibratory driving conditions, has different properties

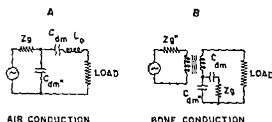



Fig 12 Electric analogues of the middle ear for air and bone-conduction driving conditions The analogues are greatly simplified and are drawn after those used by Zwislocki (1962) ($Z_g$ source impedance, air conduction, $C_{dm}$ part of the drum membrane serving transmission of energy onto the ossicular chain, $C_{dm}$ part of the eardrum attenuating energy trans[illegible] [illegible] $L$ mass of ossicular system Similar symbols are used for bone conduction) For [illegible] explanation cf text

According to the present findings the main difference is apparently in the role of the tympanic membrane In case A it acts partly as a transmitting link between the air input and the ossicular chain ($C_{dm}$), and partly as a shunt to ground ($C_{dm}$) In case B it serves as a back loading spring partly against the air cushion of the middle ear and of the ear canal ($C_{dm}$) and partly against the bone ($C_{dm}$) No attempt has been made to test out these hypotheses in an actual analogue model

*Note*—At the occasion of the recent 70th annual meeting of the Am Acad Ophthal & Otolaryng [*Transactions Am Acad Ophthal & Otolaryng* 69 797, 1965 (abstract)] Goodhill reported on clinical observations concerning surgically confirmed mallar fixation This clinical entity to a degree is similar to the experimental fixation of the tympanic membrane reported in the present paper According to Goodhill (pers comm) preoperative audiological results were similar to those caused by footplate fixation However the Carhart s notch extended to higher frequencies beyond the range of bone conduction testing of clinical audiometers i e there was a loss practically flat, for frequencies above 2000 Hz This compares well with the wide notch seen for the cat in Fig 3 of the present paper, the notch having become a flat bottomed trough extending from 250 Hz to 1000 Hz instead of the usual one centered around 600 Hz upon stapedial fixation (cf Figs 1–7)

*Acknowledgment* The authors are grateful to Mr A S Wolfe MA for conducting the clinical audiometric tests shown in Figs 1 and 2

## Appendix A

### *Measurement of the Impedance of the Middle Ear*

Acoustic impedance the unit in which measurements of the impedance of the middle ear are customarily reported is defined as the complex ratio of effective sound pressure averaged over the surface to the effective volume velocity through it The unit of measure is the acoustic *ohm* which in the cgs system is defined as dyne sec cm$^{-5}$

There are several ways most of which actually have been used by various investigators of measuring this entity For purposes of the present study the method of Møller (1960) was adopted His paper contains a brief listing of the various other methods used up to that time Møller s own method makes use of a constant current sound source which is mechanically connected to a probe microphone although care is taken for magnetic and vibratory decoupling between the two Both the transducer and the receiver are connected to a tube which is inserted into the external ear canal Care must be taken that the insert tube is sealed airtight into the ear canal Through suitable instrumentation the magnitude of the output voltage of the receiver is measured as well as the phase angle between the transducer and the receiver These voltages and phase angles

obtained for a series of frequencies, represent a measure of the impedances involved For their conversion into acoustic ohms, an electrical analogue is made consisting of an appropriate series of coils and condensors This line which is driven by a high impedance source is terminated by a variable impedance, an analogue of the impedance as seen from the tip of the tube insert (At the conclusion of the measurements on the ear it is necessary to determine the volume of the ear canal as it was when the measurements were taken and to insert equivalent values into the analogue )

The variable reactance and resistance (a condenser and resistor in parallel) are adjusted for each test frequency until the magnitudes and phase angles of the voltages at the analogue source are identical to those determined at the ear The results are then read separately in terms of the real and imaginary parts of the impedance, i e the acoustic resistances and reactances respectively

For the purposes of the present experiments, an instrument was built according to Møller's published data It proved itself satisfactory for the work on cats and dogs For the smaller species (guinea pigs and rats) however, a half-size instrument was constructed, including, of course, a corresponding analogue

It was soon found that impedance measurements are much more sensitive to fluctuations of body temperature than cochlear microphonic responses The same observation has been made by Mundie (1960 1962), Møller (1963), and Zwislocki (1963) Zwislocki when attempting to measure impedances in cadaver ears became acutely aware of this problem Low temperatures appeared to be the major source of error when the cadaver data were compared to those obtained in living human subjects It has been mentioned repeatedly in the present studies that the premedication with chlorpromazine disturbs the animal s thermo-regulation The electrical heating pad utilized heretofore which was usually connected to the power line in between test runs and disconnected during test runs, was not sufficient anymore Rectal temperatures were monitored by a thermistor couple and the derived voltages were utilized for control of the heating pad thus maintaining body temperatures approximately $\pm 0.5F°$ of the required value for each species

*Acknowledgment* The authors wish to acknowledge gratefully the advice Dr A Møller gave with regard to construction and use of the instrument as well as with respect to the evaluation of data by means of the analogue

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# IV. BONE CONDUCTION EXPERIMENTS IN ISOLATED MIDDLE-EAR SPECIMENS OF CATS

JUERGEN TONNDORF, ELLIOT C GREENFIELD,
AND ROGER S KAUFMAN

## Summary

The present experiments were planned originally for the purpose of measuring bone conduction responses in isolated middle-ear specimens of cats Although the stated objective was not achieved, the experiments provided (a) evidence for the occurrence of distortional vibrations of the cochlear capsule compatible with the concept of compressional bone conduction and (b) some information with respect to the amplitude ratio between the displacement of the stapes footplate and that of the skull when the latter is driven by a vibratory signal

## A Introduction

Results obtained in earlier studies (papers No II and III) had made it desirable to study the bone conduction responses of isolated middle-ear specimens of cats Previously, these responses had been evaluated indirectly by calculation and involved a comparison of bone-conduction responses in intact ears with those after middle-ear amputation It was hoped to obtain the displacement amplitudes of the stapes directly in isolated specimens with the aid of a so-called capacitive probe, an instrument which Békésy (1941) first introduced as an investigative tool For these purposes, he had actually adapted an instrument developed earlier by Backhaus (1930)

## B Procedure

The present authors employed an instrument of a similar design which is available commercially, the Wheathers P 655 oscillator After suitable modification, it served for such purposes in previous experiments of one of the authors (Tonndorf, 1960) It was originally designed and is marketed as part of a phonograph pick-up system Its principle is as follows The probe together with the body, the displacement of which is to be measured, forms the tuning capacitor of a high-frequency tuned circuit Any variation in the distance between these two "plates" produces a frequency

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modulation of a high frequency signal, generated elsewhere in the circuit By means of slope detection, the frequency modulation is converted into an amplitude modulation which is proportional to the initial mechanical displacement The high frequency is subsequently removed by suitable filters Since the probe is transformer-coupled to the remainder of the circuit and the transformer has a very low impedance for low-frequency signals on the side of the probe, the latter is remarkably insensitive to 60-Hz interference It therefore does not require shielding and can be made very small and light weight The obvious advantage of this instrument is that no mechanical contact is made to the system to be examined and any loading of that system is avoided

For the present purpose, the probe was fashioned from 100 $\mu$ silver wire, flattened at one end to a small plate, 0 8 mm in diameter, in order not to exceed the length of the small axis of the stapes footplate [In some preliminary experiments (cf below), ball shaped electrodes were used However, it was found that they were not sufficiently discriminating with respect to direction ] The plate was cemented onto the thinned-out end of a glass capillary, 12 mm in length, the lead-off wire running inside This length was chosen so that when the electrode was introduced through the widened internal auditory meatus into the vestibule (depth approximately 5 mm) it could be fastened precisely at its own center to the walls of the meatus in order to minimize its moment of inertia to translational motion

The probe was calibrated with the aid of a vibrator which drove a Brüel & Kjaer accelerometer (type 4308) The latter in turn faced the probe The use of $^1/_3$ octave filters (Brüel & Kjaer audio frequency spectrometer, type 2112) in the probe response line made it possible to record responses in the order of $10^{-8}$ cm (and frequently better than that)

The specimen was prepared in the following manner Immediately after sacrifice, the cat's head was split lengthwise into two halves, the brain removed, one half put into deep-freeze storage, properly protected against dehydration, the vestibule of the other half was exposed through the internal meatus The latter was widened to approximately 2 5 to 3 mm In cats, the footplate is known to be very thin and fragile and to protrude convexly into the vestibule The probe was introduced into the recording position by means of a micro-manipulator After the probe was securely fastened to the wall of the internal meatus by means of dental cement or "sticky wax", the manipulator was withdrawn Thus the probe except for its thin and flexible lead off wire was made an integral part of the specimen

## C Results

A first test was to determine with the aid of air borne signals, whether or not the probe was really capable of picking up footplate displacements

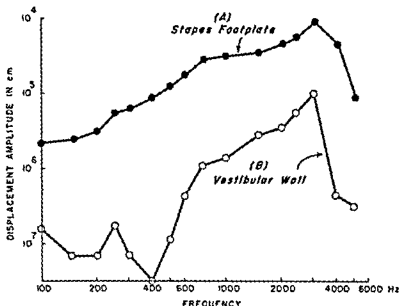



Fig 1 Response of capacitative probe (in cm displacement) to air conducted signals curve A probe facing the stapedial footplate, curve B probe facing the vestibular wall During both tests the probe was fastened to the walls of the (widened) internal auditory meatus

Fig 1 shows the response of the probe in two situations (a) facing the stapes footplate and (b) facing the lateral vestibular wall, posteriorly to the oval window The signal was monitored by a probe microphone in the external canal introduced through its osseous wall, according to a method described by Pfalz (1962) The ratio between both curves of Fig 1, minimally 19 dB at 3000 Hz and maximally 49 dB at 400 Hz, leaves no doubt that the probe when facing the footplate, was actually registering the latter's displacement relative to the surrounding bone Part of the signal picked up when the probe was facing the vestibular wall was probably still due to footplate motion especially around the resonant point of 3000 Hz where the response was rather strong (In this experiment, a ball-shaped probe was used which, as stated above, was not very discriminating with respect to direction ) In this frequency region, the two curves follow each other rather closely However, a small part might have actually been due to so called *distortional vibrations* of the bone itself, i e distortions of the internal meatus, to which the probe was fastened, with respect to the lateral vestibular wall Certainly, at an input level of 135 dB at which these readings were taken some bone responses to air-borne signals had to be expected This assumption gave rise to the next experiments

This time, a specimen was driven by vibratory signals The output rod of a bone conduction vibrator (Western Electric Model D-80904) was solidly connected t the accelerometer and the latter in turn to the skull at a point approx ately 15 mm in front and slightly above the external ear

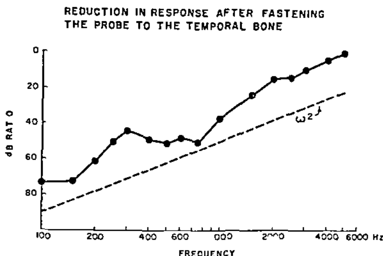



Fig. 2. Bone conducted signals, capacitative probe facing the lateral vestibular wall. Response reduction after fastening the probe to the walls of the (widened) internal auditory meatus. Originally it was not connected to the specimen but to a micro manipulator. The function $\omega^2$ is also indicated.

canal. Thus the accelerometer served to monitor the vibratory input. The skull was supported by a foam rubber cushion in a manner similar to that used in the bone conduction experiments in living animals. The probe was facing the lateral vestibular wall. Measurements were taken under two conditions: (a) with the probe remaining fixed to the micro-manipulator and (b) fastened to the rim of the internal meatus and released from the manipulator. The ratio between the two series of measurements for each frequency represents the reduction of probe to bone movement due to their fixation to each other. Fig. 2 shows the results of these measurements. The fixation of the probe was apparently quite effective for low frequencies (reduction > 70 dB), but this effect decreased rapidly, becoming zero at approximately 5000 Hz.

The question was whether or not under these circumstances a footplate motion relative to the vestibular wall and in response to vibratory stimulation could be detected at all. In the next experiment the probe was made to face the footplate. It was fastened to the rim of the internal meatus and released from the micro manipulator. Vibratory signals were employed. A first series of responses was taken with the specimen intact and a second one with the incudo stapedial joint interrupted in order to eliminate the effect of ossicular inertia. (In other specimens the stapes was removed altogether.) Results of such a pair of tests are given in Fig. 3. (Above 1000 Hz responses were too small for reliable measurements.) It is seen from Fig. 3 that there were indeed some small differences between the two sets of measurements, apparently increasing with inverse frequency. In other words, it was only in the low frequency region that the amplitude

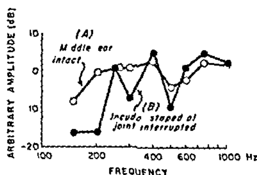



Fig 3 Responses of the capacitative probe to bone-conducted signals, capacitative probe facing the stapedial footplate Curve A middle ear intact, curve B incudo stapedial joint interrupted

of stapedial displacement was larger than that of the surrounding bone as seen by the probe.

Attempts were made to employ two probes in a differential manner, one facing the footplate and the other one the lateral vestibular wall, in an effort to separate one type of motion from the other However, these attempts failed, because it was not possible to isolate the two probes and the associated FM circuits from each other electrically, while still maintaining the necessary close spatial relationship

In a last experiment, the present method was utilized to measure the amplitude ratio of the displacement at the point of vibratory input to the skull and that of the lateral vestibular wall for various frequencies The point of input was once more 15 mm in front and slightly above the external ear canal The probe was facing the lateral vestibular wall, but was supported independent of the skull

Fig 4 gives two sample recordings, one for a skull supported in the usual fashion upon a foam rubber cushion (curve A), the other with the skull fastened only to the vibrator assembly, upon which it was actually balanced (curve B) In most specimens and in both of the above instances, there was a slight transmission loss, as a rule slightly higher when the specimen was supported by the vibrator and slightly less when it was supported upon foam rubber, as is typically shown by the two samples of Fig 4 Furthermore, it was found that the transmission through the skull was not independent of frequency The form of the curve varied both with the specimen and with the mode of support in a seemingly uncontrollable manner Curve B of Fig 4 is an example of a fairly flat curve More often, there were sharp variations such as those around 300 Hz of curve A The variations were not confined to any particular frequency, altered profoundly when the support was changed only slightly, and often reached, and sometimes exceeded $\pm 10$ dB

It is well n a n in clinical audiometric testing that the varying amount

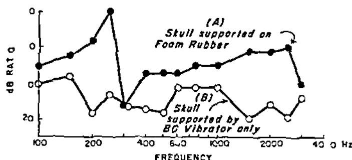



Fig 4 Displacement ratio lateral vestibular wall (capacitative probe) *vs* skull at bone input (accelerometer) Curve A specimen supported on foam rubber in a manner similar to that used with living animals in other studies curve B specimen supported by the bone-conduction driving system only

of soft tissues interposed between the vibrator and the skull and their variable consistency introduces some response variations (Békésy 1932 Barany 1938 and Konig 1955) This factor had of course been excluded from the present experiments as well as from the preceding experiments in live animals Hence it had been tacitly assumed that monitoring the vibratory input at its point of application provided an accurate measure of the energy eventually reaching the ear Fig 4 shows that this assumption is only approximately correct and that bone conduction monitoring may be less reliable than monitoring the input of air borne signals to the ear by means of a probe microphone in front of the tympanic membrane

This finding appears to explain the differences in smoothness between individual air and bone conduction curves which were taken in live animals in a later study with the aid of cochlear microphonic responses (paper No VI Fig 2) Both inputs were monitored The air conduction response curves were consistently smoother than the bone conduction response curves Thus the variation could not be caused in the receptor organ It had been found earlier (paper No I) that as long as the support of the animal s head and its connection with the vibrator were not altered bone conduction response curves were well reproducible in repeated trials That the variations were essentially random and thus virtually beyond control in the present experiments is indicated by the fact that bone conduction responses became considerably smoother when averaged over a number of animals (paper No VI Figs 2 and 3) The smaller the animal the more the bone conduction responses (in terms of cochlear microphonics) were found to be influenced by head position and support Standardization of the procedure was found most difficult in rats (cf paper No III) By the same token it is quite possible that the difficulties encountered here are of lesser importance in human subjects when the head is held erect and is free of any outside support and the vibrator is fastened upon the head itself as it is customarily done

## D Discussion

The original objectives of the experiments were not achieved Nevertheless, the results obtained gave some other information (a) with respect to the occurrence of compressional bone conduction and (b) with regard to the relative magnitude of the ossicular response to vibratory stimulation of the skull

(a) *Compressional bone conduction*—Fig 2 indicated that as frequency went higher the fixation of the probe to the internal meatus and relative to the vestibular walls became more and more ineffectual Above 5000 Hz, it had no effect at all If this result were merely due to vibrations of the probe around its point of fixation, one would expect a clear-cut minimum at the resonant point of the probe By slight tapping of the probe this point was assessed to be approximately in the vicinity of 300 Hz In Fig 3 there was indeed a relative maximum at 300 Hz, but otherwise the curve approximated a slope of 12 dB/octave, i e it varied with the square of frequency In other words, whatever was causing the fixation to become gradually ineffectual, increased with the square of frequency These findings suggested that it might well be *distortional vibrations* of the entire specimen, especially between the various parts of the vestibular and the internal meatus (and including the probe) which rendered the fixation of the probe gradually ineffectual Distortional vibrations, of course, are the *prerequisite of compressional bone conduction* Significantly, it will be shown in paper No VI (Fig 10), that in the range below 1000 Hz the pure compressional component varies approximately with the square of frequency

Another piece of evidence for the existence of distortional vibrations (and thus for compressional bone conduction) was seen earlier It was reported in paper No I that the occlusion of the cochlea aqueduct in conjunction with that of the round window (Fig 9, loc cit ) affected only bone conduction responses, but not those due to air-conducted signal This finding had also been interpreted as evidence for the generation of distortional vibrations of the cochlear capsule in response to vibratory stimulation of the skull

(b) *The relative magnitude of ossicular responses to vibratory stimulation of the skull*—Fig 3 had shown that the amplitude of the footplate displacement and that of the surrounding bone when responding to vibratory stimulation might be approximately equal to each other in the range from 400 Hz to 600 Hz According to Fig 2, the fixation of the probe was still effective in this region in minimizing relative displacements between the probe and the vestibular walls Since (i) the reduction of pick up by the probe due to its fixation to the internal meatus had amounted to approximately 50 dB in the range in question, (ii) there was an average transmission loss of about 10 dB between the surface of the skull and the region of the ear (Fig 4) and (iii) the amplitude of the stapes foot-

plate was approximately of the same magnitude as that of the oval window frame as seen by the probe (Fig 3), it stands to reason that the *amplitude of the stapes footplate relative to the surrounding bone and due to the ossicular inertial response, might be in the order of* *ɔ0 dB to −70 dB in reference to the vibratory input* Due to the uncertainties of the present estimate it appears preferable to give a rather wide range

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# V THE OCCLUSION OF THE EXTERNAL EAR CANAL ITS EFFECT UPON BONE CONDUCTION IN CATS

JUERGEN TONNDORF ELLIOT C GREENFIELD
AND ROGER S KAUFMAN


## Summary

The occlusion effect of the external ear canal with respect to the bone conduction response is a complex phenomenon

(1) The walls of the external canal when vibrating radiate acoustic energy into its lumen from where part of it is transmitted toward the receptor organ *via* the middle ear In this respect the ear canal when open constitutes a high pass filter Hence its occlusion produces a low frequency emphasis In cats this particular effect is most dominant below 600 Hz which represents the resonant point of the cat s middle ear for bone conducted signals

(2) The air contained in the external canal constitutes a load upon the tympanic membrane Thus any changes of the resonant properties of the canal due to a modification in length its occlusion or both will alter the *load effect In cats this partial effect is most important in the middle to* high frequency region

Therefore the second effect influences the middle ear responses to bone-conducted sounds directly while the first one requires a functional middle ear for its proper transmission

Deep seated occlusions leaving small air spaces in front of the tympanic membrane attenuate the bone conduction contribution of the external canal both at the source (reduction of the radiating wall space) and in transmission (loading of the tympanic membrane) thus eliminating the occlusion effect and ultimately even reducing the bone conduction responses

In man the radiation of acoustic energy into the ear canal may be aided by the out of phase motion of the lower jaw as first described by Bekesy However this latter effect appears to play a minor role in the total phenomenon


## A Introduction

Politzer in his *Geschichte der Ohrenheilkunde* (1913) credited Tortual of Germany and Wheatstone of England for having independently dis

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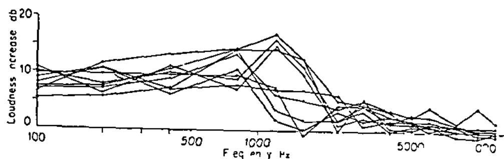



FIG 1 Loudness changes due to the occlusion of the external ear canal (after Békés 1939)

covered in 1827 what later became known as the test of Weber (1834) Frey in his article on bone conduction in Politzer's book (vol 2) preferred to call it the *Weber Wheatstone* test

This test is based upon the observation which has since become well known that impairments of middle ear function produce an *improvement of bone conduction* responses in the involved ear If the underlying condition exists in one ear only it leads to a distinct *lateralization* of the perceived sound toward that side

The diversity of opinions concerning the mechanism underlying this test stems from an unfortunate set of circumstances It so happens that almost all impairments located anywhere along the conductive pathways of the ear from the entrance to the external ear canal to the stapes footplate produce essentially similar changes in bone conduction responses Consequently many writers felt compelled to search for a single explanation that would cover *all* observational facts Since this objective was never achieved for reasons which will become apparent in the present paper experienced audiologists such as Hirsh (1952) Fournier (1959) and Carhart (1962) voiced their scepticism concerning the compatibility of theory and practice in this regard

The actual objective of the present study which was conducted in laboratory animals (cats) was the *occlusion effect of the external ear canal* In Appendix A attempts will be made to explain the phenomena underlying the clinical tests of *Bing Runge Weber* and *Gellé* These explanations will be based upon the results of the present study and also upon those of previous ones (papers I and II)

Occlusion of the external ear canal as is already known produces a low frequency emphasis for bone conducted signals (Békésy 1932 1939 Onchi 1954 Allen & Fernandez 1960 E H Huizing 1960 and others) Fig 1 shows the increase in loudness in one ear for bone conducted signals upon occlusion of its external canal according to Békésy (1939) In first approximation the increase is seen to be proportional to inverse frequency Some writers have also called attention to the fact that there are often

reductions in bone conduction responses in the middle and high-frequency regions (Watson & Gales, 1943, E H Huizing, 1960, Groen, 1962)

Rinne (1855) thought that the occlusion effect could be explained on the basis of altered resonances in the external canal Huizing (1960) revived this concept and at the same time put it on a sounder scientific basis

The explanation which was most widely accepted for a long period of time, was that of Mach (1874), the so-called "Outflow Theory". This author had ingeniously reasoned that "if air-borne sound enters the cochlea easily by virtue of the function of the middle-ear structures, one may assume that it will leave the cochlea in a reverse direction with equal ease". This is a legitimate application of the Reciprocity Theorem of Helmholtz to the problem of the middle-ear transformer In Mach's time it was widely believed that vibratory energy would reach the cochlea exclusively by the osseous route (compressional bone conduction, in modern terminology). Consequently, Mach thought that the middle ear might constitute a leak through which some of the applied vibratory energy might escape ("outflow") so that the receptor organ would receive less energy than originally transmitted to it An impairment of this physiological "outflow" route, while the "inflow" was kept constant, would retain a larger amount of energy for reception by the cochlea A great number of mostly earlier experiments, such as those of Politzer (1864), Lucae (1864), Claus (1909), Runge (1923), W Tonndorf (1924), and others, seemed at first sight to support Mach's notion Allen & Fernández (1960) might be considered modern proponents of Mach's Theory

Most of the experimental "proof" in favor of Mach, submitted by the above authors, rests upon the following observation A microphone or similar device was brought to the entrance of or even into the external canal On vibratory stimulation of the skull, this microphone registered a certain sound pressure which was thought to be due to displacements of the tympanic membrane and hence considered proof for the radiation of acoustic energy from that membrane Barany (1938) first pointed out the fallacy in this argument Such devices *cannot exclusively register the* displacements of the tympanic membrane in reference to the external ear canal The entire skull, external ear canal and tympanic membrane included is displaced relative to the microphone, which has a frame of reference different from that of the vibrating skull From data so obtained, one cannot extricate the separate displacement of the tympanic membrane in reference to the external canal Barany rightly called this type of experiment the "classical error of Bone Conduction Theory"

Previous experiments in the present series (Tonndorf & Tabor, 1962, paper No I) had given evidence for a slight leakage of compressional bone conduc[illegible] through the oval window In line with the original concept of Herzog [illegible]z (1926) this leakage was found to depend upon the yielding of the st[illegible] plate It was best demonstrated when the middle ear was absent Sin[illegible] vibratory stimulation the middle ear is not a passively

transmitting system, as Mach had thought but is contributing actively and significantly to the overall bone conduction response (ossicular inertia, Bárány, 1938), it ordinarily obscures the oval window leakage completely. In an intact ear, the latter effect plays a rather insignificant role. Although it has some similarities to Mach's outflow concept, the finding of the oval-window leakage should not be considered a confirmation of Mach's theory.

Guild (1936) was of the opinion that the occlusion effect was due to the elimination of masking of bone conduction signals which is normally exerted by air-borne sound.

Békésy (1941) made the following observation. When the skull is subjected to vibrations, the lower jaw follows this motion, but with different amplitudes and phases, because of its different size and its loose coupling to the skull. Since the head of the mandible borders on the cartilaginous portion of the outer ear canal, a relative motion set up at this point must affect the shape of the canal in synchrony with the applied signal. With the ear canal occluded, these changes in shape should lead to proportional pressure changes, which in turn are transmitted to the inner ear via displacements of the tympanic membrane and the ossicular chain. Békésy saw further support for his concept in the finding that a plug inserted deeply into the osseous portion of the ear canal, i.e. beyond its soft compressible outer portion, did not produce the usual improvement in bone-conduction hearing.

Some counter evidence against Békésy's concept was presented by Allen & Fernandez (1960). These authors had occasion to examine the occlusion effect in two patients with the lower jaw missing on one side. It turned out that in both of these patients the occlusion effect could be elicited equally well in both ears. Reger (pers. comm.) has observed another such case.

However, Békésy's concept cannot be dismissed that lightly. *Franke et al.* (1952) have measured in detail the phase relationships between the skull and the lower jaw for various *frequencies under bone conduction driving* conditions. They confirmed Békésy's *original concept in this regard* and have given some further evidence indicating that the *lower jaw really contributes* to the occlusion effect.

Other hypotheses, notably that of *Groen (1962)*, which have bearing upon the phenomenon of the Weber test, will be dealt with in *Appendix A*.

### B. *Procedure*

Cats were used in the *present experiments. The method of anesthesia, a* combination of chlorpromazine *and sodium nembutal, has been described* earlier (cf. paper No. I). *Cochlear microphonics served once more for* registration of *cochlear responses to bone-conduction signals. Since no* manipulation of the *cochlear windows were contemplated, a single round*-window electrode *was utilized (100 μ enameled silver wire), its tip*

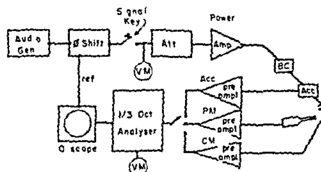



Fig 2 Experimental arrangement (block diagram) for recording (a) cochlear microphonic responses (CM), (b) sound pressure levels in the external ear canal by means of a probe microphone (PM), both in response to (c) bone-conducted signals monitored by an accelerometer ($A_{cc}$) Voltage amplitudes were read as well as phase angles relative to the electrical signal

fashioned into a small ball The small opening in the bulla wall, necessary for the introduction of the electrode, was sealed with dental cement A reference electrode was placed into the neck muscles

A block diagram of the experimental arrangement is given in Fig 2 Bone conduction signals were delivered by the same vibrator used previously (cf papers I to III) Western Electric, Model D-80904 However, a newly available, small and improved accelerometer (Bruel and Kjaer, type 4328), with an extended frequency range up to 25 kHz, made it possible to integrate the driving and monitoring systems The bone conduction vibrator was solidly fastened to the accelerometer and the latter in turn onto a ⊥ shaped screw which had been secured to the bone by means of a nut and washer in the temporal region of the skull through an extra-dural opening The screw was located approximately 1 5 cm in front and slightly above the external ear canal in order to leave the latter unobstructed The entire bone conduction driving and monitoring system was suspended from above to reduce its weight and to improve its frequency response By means of this arrangement, it was possible to monitor the bone-conduction input *directly* in contrast to the *relative method used earlier* (cf papers I to III however with regard to the uncertainties still adherent to this method cf papers IV and VI)

The head of the animal was on its side, held loosely in place by a Tschermak head holder, with the test ear facing upward The entire animal was supported on a thick foam rubber cushion.

Except for a large grounded metal plate underneath the animal the entire experiments were done without any screening to facilitate easy access for the necessary manipulations Yet judicious attention to ground connections gave low noise levels (8 to 10 µv, broad-band amplification) A 1/3 octave sound analyser Bruel and Kjaer, type 2112) in the response line r … levels to approximately 0 5 µv so that responses were … the 5 µv level … S/N ≈ 20 dB)

… und pressur … ls within the external ear canal, caused

by the vibratory stimulation were obtained with the aid of a probe microphone (Bruel and Kjaer, type 4134), equipped with a probe 40 mm long and 1 mm in diameter, of the UA 0040 probe kit) The tip of the probe microphone was inserted with a snug fit through a small hole drilled into the antero inferior lip of the osseous ear canal perpendicularly to the plane of the tympanic membrane according to a method described by Pfalz (1962) A polyethylene sleeve over the probe permitted its tip to extend approximately 0 5–1 mm into the ear canal, lying less than 1 mm in front of the tympanic membrane Its position perpendicular to the tympanic membrane, was the same in which the probe had been calibrated by means of the Bruel and Kjaer probe kit

All voltage readings (accelerometer, probe microphone and cochlear microphonic responses) were taken through the same Bruel and Kjaer band analyser mentioned above (cf Fig 2) All phase readings (accelerometer, probe microphone, and cochlear microphonic responses) relative to the electrical input to the driving system were taken with the aid of a phase shifter (Grason Stadtler, type 533) in the manner indicated in Fig 2 From data so obtained any desired phase relationship could be computed (For further information concerning the way phase relationships were defined and computed from the present data cf paper No VI appendix B )

During the preparatory surgery the pinna of the test ear was amputated leaving a short stump of the cartilaginous canal, approximately 1 5 cm in length A small ear speculum of appropriate size was tied into the latter Various lengths of fairly thick walled polyethylene tubing (#380) were inserted into the speculum in order to simulate variations in lengths of the ear canal Good fit of the speculum as well as of the inserts was assured by the use of 'sticky wax' and a hard petroleum jelly The ear canals were occluded by a cap snugly fitting over the inserts The cap was equipped with a small air vent for proper equilibration of pressure The vent in turn was closed by a small solid plug as soon as the cap was set in place This precautionary method had originally been advocated by Bekesy (1941) The speculum could also be occluded by a deep fitting solid insert (also equipped with an air vent) which reduced the air space in front of the tympanic membrane to its smallest value (cf below)

A series of eight animals served for various pilot experiments From their results a test sequence was developed which was then followed in a second series of four animals Since a complete set of data was obtained from each animal of the latter series this group facilitated ready comparison between various test results in the same animal Most of the results reported in the following stem from the second series of animals

## C Results

Fig 3 shows the change in cochlear microphonic responses including both amplitude and phase due to a deep occlusion of the ear canal in a series

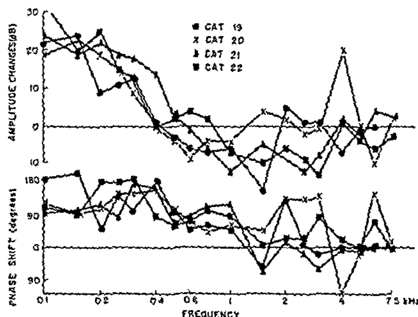



Fig. 3. Amplitude changes and phase shifts of cochlear microphonics in response to bone conducted signals, caused by occlusion of the external ear canal. Length of the ear canal 3.5 cm, insert plug at 2 cm from the plane of the tympanic membrane. Individual results from four animals for 18 frequencies in the range from 10 Hz to 7500 Hz. (Note the aberrant curve for cat #20 in the high frequency region.) It is noted that a few phase values (e.g. for animals #19, at 100 Hz and 150 Hz) have been plotted as being slightly larger than +180°. This is a reasonable correction in plotting. Actually, phases in order to show average trends, should be given on a cylindrical plot so that ±180° fall on the same point.

of four animals. The length of the "ear canal", before occlusion, was 3.5 cm. The plug was inserted to within 2 cm in front of the tympanic membrane. There was a noticeable improvement of responses in the low frequency region (Fig. 3 top), below a cut-off point of approximately 600 Hz, and in some proportion with inverse frequency. The between-animal variations in this region were reasonably small, above 600 Hz they were considerably greater. In the middle frequencies, there was some reduction of responses, varying in magnitude and in the extent of the frequency range involved. At higher frequencies, changes again became smaller. The deviant changes in animal #20, especially the sharply limited improvement around 4000 Hz, will be commented upon later. Taken as a whole, the changes of Fig. 3 were not unlike those seen in human subjects (cf. Fig. 1).

Positive phase shifts (Fig. 3 bottom), accompanied the amplitude gains in the low frequencies, again with small variations between animals. In the higher frequencies (above 1000 Hz), variations once more became greater, the sharp deviation at 4000 Hz for animal #20 being especially noted.

The next two figures (Figs. 4 and 5) show the effects of varying the length of the ear canal both in the open and the occluded state. Results of

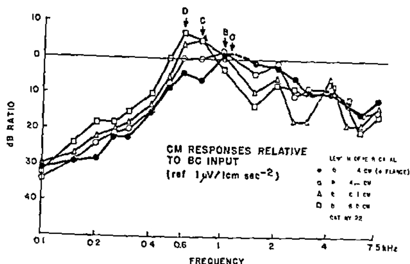



Fig 4 Cochlear microphonic responses relative to the bone conduction input (references 1 μV/1cm sec⁻²) for 18 frequencies between 100 Hz and 7500 Hz with the ear canal *open* parameter length of the ear canal Data from one individual animal

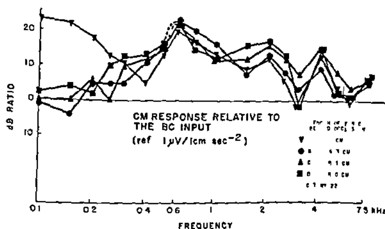



Fig 5 Cochlear microphonic responses relative to the bone conduction input (reference 1 μV/1 cm sec⁻²) for 18 frequencies between 100 Hz and 7500 Hz with the ear canal *occluded* parameter length of the ear canal when occluded Data from one individual animal (same as Fig 4)

only one animal are given since averaging results over several animals tended to obscure the sharp variations of individual curves in the higher frequencies *In both figures* the ratio of the cochlear microphonic responses to the bone input are given with 1 μV/1 cm sec⁻² as the reference First of all it is noted that the curves of Fig 5 as a whole lie consistently higher than those *of Fig 4 In* other words the improvement of the responses upon occlusion of the ear canal shown in Fig 3 for the example of one particular length of the canal occurred with all other lengths

Closer inspection of Fig 4 indicates a small, but systematic shift of the response curve toward the left, i e toward lower frequencies, with increasing lengths of the ear canals. This shift is most clearly seen at the frequencies at and below the respective resonant points but can also be recognized at higher frequencies

In Fig 5, with the ear canals occluded, the systematic shifts in position of the response curves are not so readily apparent The shifts can be recognized between 400 Hz and 600 Hz and somewhat less clearly above 600 Hz Below 400 Hz, they are still systematic for inserts *B'*, *C'* and *D'* However, the curve pertaining to insert *A'* runs an independent course of its own

It is clear from Figs 4 and 5 that a variation in length of the open ear canal affects its resonant properties as one would expect, i e the resonance point varies with the inverse length of the canal It is also clear that this variation is reflected in an appropriate change of the microphonic responses Although this fact is less apparent when the ear canal is occluded, one gains the impression that changes in the resonant properties of the external canal might well play a role in the occlusion effect as originally suggested by Rinne (1855) and by Huizing (1960)

W Tonndorf (1924) had performed similar experiments in human subjects obtaining similar results in which he saw support for Mach's "Outflow Theory" It will be shown later that his conclusions are not the only ones possible and that, in fact, there is some positive evidence against Mach's Theory

In the present experiments, sound pressure levels in the external ear canal were not registered, of course, to "prove" displacements of the tympanic membrane The microphone merely served the purpose of determining whether there might be a correlation between the changes in microphone outputs and those in cochlear microphonic responses under the various experimental conditions Two sample comparisons, for an ear canal of 4 3 cm in length, open and occluded, are shown in Fig 6 The data are given once more, for a single animal Both sets of curves (microphone and cochlear responses) are referred to the bone input in an identical manner The graphs are limited to frequencies below 1500 Hz because at higher frequencies the probe microphone required such large bone inputs that the cochlear microphonic responses became nonlinear, making the value of the comparison questionable

Fig 6 shows that under both conditions (ear canal open and occluded the two curves representing the probe microphone responses and those of the cochlea can almost be superimposed upon each other Furthermore, comparison of the top and bottom of Fig 6 indicates that the occlusion of the ear canal improved the cochlear response and the output of the microphone approximately to equal degrees

Th[illegible] [illegible] rrelation between the two kinds of curves in Fig 6 requires [illegible] furt[illegible] [illegible]ment since the original cochlear microphonic data had

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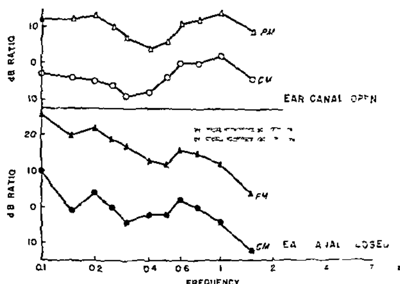



FIG 6 Comparison between probe microphone readings in the [illegible] cochlear microphonic responses (corrected by subtracting the resp[illegible] ear under bone-conduction stimulation) Data from one individual anim[illegible] frequencies between 100 Hz and 1500 Hz ear canal open (top) and occluded [illegible]ttom

to be converted for the purpose of this comparison Ordinarily, the cochlear response curves, as was shown in Figs 4 and 5 have a steep positive slope below 600 Hz This is due to the fact that the middle ear of the cat has its resonant point at approximately 600 Hz for bone conducted signals (cf papers I, III, and VI) Below this point, the slope of the bone conduction response curve is almost exclusively determined by the middle ear Hence the cochlear microphonics "read" the sound pressure in the external canal (if one assumes that is what they are doing) through the middle ear system whereas the probe microphone reads it directly For purposes of the present comparison, therefore, the cochlear microphonic readings were corrected by subtracting the middle ear responses (cf paper No VI) Fig 7 may be considered in further justification of this correction It gives the ratio between cochlear microphonic responses to probe microphone outputs for equal bone inputs in seven different test situations (open and occluded ear canals of various length) in a single animal (The curves are superimposed upon one another to show that they are all governed by the same factors ) The sharp discontinuity of the curves at 600 Hz is noted Below this point, the slope is proportional to the square of frequency which happens to be approximately the slope of the middle-ear response curve for bone conducted sounds (cf paper No VI) The narrow distribution of the curves in Fig 7 indicates that the relationship of Fig 6 the original data of which were included in Fig 7, existed for other experimental situations as well

Returning once more to Fig 6, one can easily derive the following three conclusions (1) When the skull is subjected to vibratory stimulation,

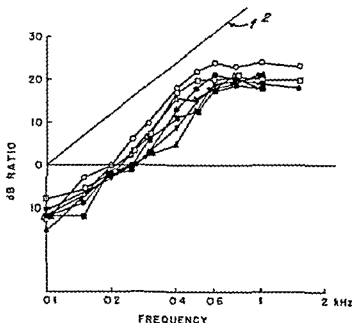



Fig Ratio between the cochlear microphonic responses and the probe microphone readings (arbit reference) for 11 frequencies between 100 Hz and 1500 Hz under bone conduction stimulation Each of the seven superimposed curves represents one experimental situation (open and closed ear canals of various length) Data from the same animal shown in Fig 6

occlusion of the external ear canal produces changes in sound pressure registered therein (2) The magnitude of these changes is readily reflected in alterations of the cochlear microphonic responses (3) The shape of the cochlear response curves (provided the response of the middle ear has been subtracted) resembles that of the sound pressure curves recorded from the external canal for various experimental situations Before the significance of these conclusions can be discussed further a number of additional observations must be reported

Bekesy (1941) as was already mentioned had explained the occlusion effect on the basis of the out of phase motions of the lower jaw In cats the structural relation between the lower jaw and the external ear canal is quite remote The mandibular capitulum does not border on the cartilaginous portion of the external canal as it does in man And yet, the occlusion effect produced in this species (Fig 3) was strikingly similar to that commonly seen in man (Fig 1) To test this point further the mandible was surgically resected in two animals and the occlusion effect determined for various lengths of the ear canal before and after surgery A comparison of Figs 8 and 9 indicates that, although there are some minor differences between the two sets of curves (mainly concerning insert C in the high frequency region) the general shape of the curves remained [illegible] P [illegible] man *et al* (1965) reported the same negative findings [illegible] eir own series of experiments This experiment indicates that the

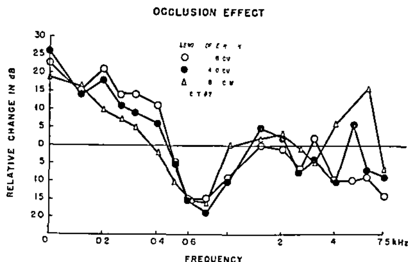



Fig 8 Occlusion effect for ear canals of various length in one individual animal mandible intact

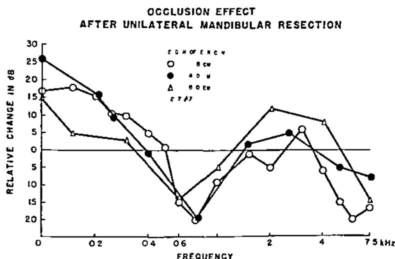



Fig 9 Occlusion effect for ear canals of various length in the same animals as shown in Fig 8 after surgical resection of the mandible on the side of the test ear

lower jaw effect which Franke *et al* (1952) had substantiated must play a *minor* role in the occlusion phenomenon

The occlusion effect was found to depend upon the condition of the tympanic membrane In a different experimental series (paper No VI) two animals having a subacute otitis media were accidentally included The tympanic membranes were closed and thickened, but not bulging The mucous membranes were many times their normal thickness to the point of being almost polypoid in some areas The occlusion effect was not demonstrable in either of the two animals *These accidental observations* are in line with clinical observations in human patients

In one of the animals shown in Fig 3 (#20) the occlusion effect could

not be elicited at first The animal in question was a large tom-cat (3 5 kg), much larger than those usually preferred by the present authors At no time was the anesthesia really satisfactory. The animal was never completely relaxed, either during the preparation or during the testing period. Upon occlusion of the external canal, there were the usual changes in probe microphone readings, but no alterations of the cochlear microphonic responses whatsoever. The bulla was then re-opened, the tendon of the *tensor tympani* muscle severed at its point of insertion at the malleus, and the bulla re-sealed Thereafter, the results shown in Fig. 3 were obtained. They were quite similar as to magnitude and frequency ranges to those observed in the other animals. The only major difference observed occurred at [illegible]000 Hz (cf Fig 3). The latter deviation was found for *all* experimental situations It was therefore assumed to be related to the altered suspension of the middle-ear ossicles after severance of the tendon of the *tensor tympani* muscle This accidental finding (together with the observations on the two animals with otitis media) elucidated one important point, namely that a ***compliant tympanic membrane is essential for the successful demonstration of the occlusion effect.***

The routine insertion of the probe microphone into the external canal for the purpose of monitoring air-borne sound generated by the vibrations of the skull facilitated a relatively simple test of the validity of Mach's Outflow Theory. It is recalled that previously such readings had been taken in support of this theory

If one supposes Mach and his supporters to be ri iption
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should expect that the removal of this transduc ed
detrimental effect upon the sound pressure re
Even if the tympanic membrane were not the
tenuation would be bound to occur as the r
Fig 10 shows the changes in sound pressure le
three animals, and the shifts in phase resulting
tympanic membrane There were practically no
frequencies below 1000 Hz, either in amplitud
*changes were conspicuously absent in the freque*
*occlusion effect is known to be most prominent*
the small and non-systematic changes observed a
following explanation is offered After removal of t
the acoustic properties of the cavity the micropho
altered Because of the small dimensions involved,
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The outcome of this simple experiment is cons
against Mach's Theory The counter-evidence of Bárá
correct as it is, must be regarded as *circumstantial* e

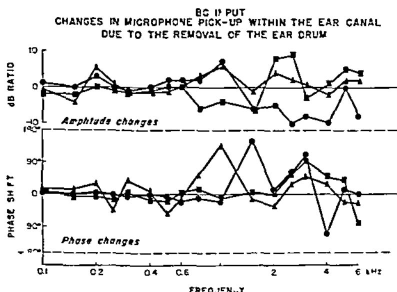



Fig 10 Changes in sound pressure levels (top) and shifts in phase (bottom) in the external ear canal due to the removal of the tympanic membrane. Bone-conduction stimulation. Individual results from three animals

The ossicular inertial response of the middle ear of Bárány (1938) the existence of which was confirmed in paper No I requires some displacement of the tympanic membrane relative to the ear canal. The reasons that these vibrations were not detected by the present method lies in the same difficulty encountered earlier in paper No IV. Their amplitudes are extremely small compared to those of the bone. The estimate made in paper No IV placed the displacement ratio ossicular chain (stapes)/bone in the low to middle frequency region approximately at −50 dB to −70 dB

D Discussion

1 *Masking Effects*

Guild (1936) as is recalled had voiced the opinion that the occlusion effect might be caused by the exclusion of masking of bone-conducted signals which is normally exerted by air borne sounds. There is no doubt that this may occasionally be a contributory factor. However the present evidence suggests that the exclusion of masking cannot be the *sole* explanation of the observed effect with its characteristic frequency-dependent changes (cf Figs 1 and 3). First of all the present experiments were done under relatively quiet conditions. Secondly the changes observed concerned cochlear microphonic responses and it is well known that masking does not affect cochlear microphonics but is an interference taking place at higher levels of the auditory system

*) (c i Resonances*

In )der to examine more closely the potential contribution of ear canal r s nances to the occlusion effect the data of Figs 4 and 5 were replotted is r sponse levels *vs* length of the car canal the latter being normalized for the w ivclength of each test frequency Fig 11 shows the results in ne inimal for a number of frequencies separately under both conditions wi l the ear canal open and (b) occluded

It must be mentioned here that Huizing (1960) employed essentially t ame approach as that used in Fig 11 in an effort to demonstrate that u occlt ion effect should be explained as resulting from a change in i ant properties of the external ear canal However Huizing s considera i were limited to the middle frequencies

I a the *middle frequencies* where the *middle ear impedance is relatively low* the *open* ear may be *considered in first approximation a tube open* at *b th* ends and the *occluded* ear canal one *open* at *one* end only The well known rules governing the acoustic properties of such tubes (cf for example Beranek 1954) lead one to expect the occurrence of a pressure minimum at the drum membrane for $^1/_2 \lambda$ with the ear canal open and a pressure maximum at the same point with the ear canal occluded The drum membrane at least at frequencies below its resonant point is known to be a pressure receiver (Bekesy, 1941 *a*) The curve sections which hap pen to be in the right range of wavelength (i e 2 5 and 3 0 kHz) support the above proposition in both situations The others, as far as their extra pol itions indicate at least do not contradict it

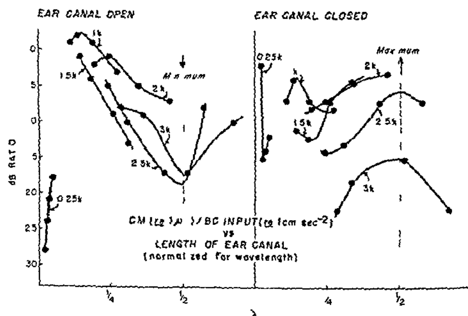



Fig 11 Data of Fig 4 an rep ot l as esponse levels (cochl microphon cs/bone-conduct n n p r se s length of the ear canal (normalized for w a elength Left al op n r ght ar canal occluded

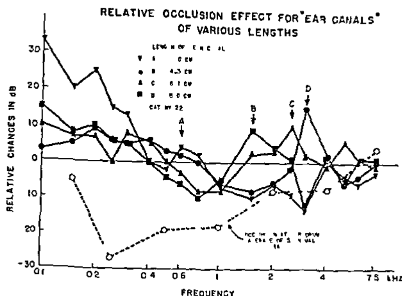



Fig 12 Changes in microphonic responses due to the occlusion of the ear canal under bone-conduction stimulation Data from one single animal 18 frequencies between 100 Hz and 1500 Hz, four different lengths of the ear canal (A through D)

The dotted line refers to a different set of data (from paper No I average of five animals) Here, the occlusion had been affected directly at the level of the tympanic membrane, laying no air space between the latter and the insert (Seven frequencies between 150 Hz and 7500 Hz)

In the case of low frequencies (the sole example of 250 Hz in Fig 11), the middle-ear impedance is much higher It may therefore be more appropriate to consider the open ear canal a tube open at one end only, and the occluded canal one closed at both ends In the first case, a pressure maximum should occur at the tympanic membrane for $^1/_4$ λ, and in the latter one for $^1/_2$ λ As far as the open ear canal is concerned, this proposition is at least not contradicted by the small curve section shown at the extreme left of Fig 11 However, with the canal occluded the rule applies only partially at best Particularly, the lowest point (corresponding to insert *A'*, cf Fig 12) does not fit the rule at all and it is at the low frequencies, as is recalled that the occlusion effect was most pronounced

Fig 12 gives the amplitude changes due to the occlusion of the ear canal for various frequencies and for ear canals of various lengths, once more for the case of one individual animal It is obvious from their systematic order that the sharp peaks (labelled by arrows) and the dips which are not quite so sharp in the middle to high frequencies, are due to altered resonant properties of the external canal in the sense of Fig 11 The following questions then arise (1) What is responsible for the effect at lower frequencies and (2) How does the loss produced by an occlusion made directly at the level of the tympanic membrane (cf Fig 12) fit into the picture?

2 *Canal Resonances*

In order to examine more closely the potential contribution of ear-canal resonances to the occlusion effect, the data of Figs 4 and 5 were replotted as response levels *vs.* length of the ear canal, the latter being normalized for the wavelength of each test frequency Fig 11 shows the results in one animal for a number of frequencies separately under both conditions (a) with the ear canal open and (b) occluded

It must be mentioned here that Huizing (1960) employed essentially the same approach as that used in Fig 11 in an effort to demonstrate that the occlusion effect should be explained as resulting from a change in resonant properties of the external ear canal However, Huizing's considerations were limited to the middle frequencies

In the middle frequencies, where the middle-ear impedance is relatively low, the *open* ear may be considered, in first approximation, a tube *open* at *both* ends, and the *occluded* ear canal one *open* at *one* end only The well-known rules governing the acoustic properties of such tubes (cf for example, Beranek, 1954), lead one to expect the occurrence of a pressure minimum at the drum membrane for $^1/_2\ \lambda$ with the ear canal open, and a pressure maximum at the same point with the ear canal occluded The drum membrane, at least at frequencies below its resonant point, is known to be a pressure receiver (Békésy, 1941 *a*). The curve sections which happen to be in the right range of wavelength (i e 2 5 and 3 0 kHz) support the above proposition in both situations The others, as far as their extrapolations indicate, at least do not contradict it

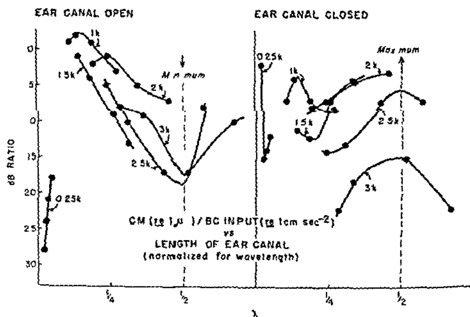



Fig 11 [illegible] replotted as response levels (cochl microphonics/bone-conduction input ref [illegible] sec ) vs length of the ear canal (normalized for wavelength) Left ear canal open right ear canal occluded

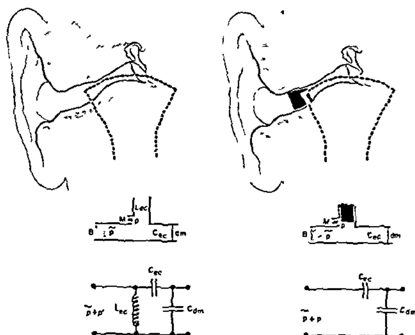



FIG 13 The occlusion effect of the external ear canal Anatomical drawings and their mechanical and electrical equivalents Left side ear canal open, right side ear canal occluded

In the anatomical drawings, the mandible is given as a dotted outline, because it lies in front of the plane of the drawing

In the mechanical equivalent (left side), the vibrating bony walls are given as a piston (*B*) which produces an alternating pressure (*p*) The mandible (*M*) represents an auxiliary pressure source (*p'*) The air in the external canal terminated by the drum membrane (*dm*), represents a compliance ($C_{ec}$) and has a leak ($L_{ec}$) toward the outside, the external opening The latter is eliminated when the canal is occluded (right side)

The electrical equivalent (left side) with its inductive shunt to ground ($L_{ec}$) is recognized as a high pass filter Occlusion of the shunt (right side) eliminates the filter effect The two capacitances ($C_{ec}$) and ($C_{dm}$) are arranged according to the electric analogue of the middle ear by Zwislocki (1962), but in a simplified manner

the low-frequency emphasis was small It was only the deep insert within the ear speculum (cf procedure) which occluded the ear canal optimally

When the plug insert is gradually moved deeper into the osseous ear canal two things must be expected to happen (1) The surface area of the ear canal radiating sound into its own lumen must become smaller (2) As the air cushion in front of it decreases in volume, the tympanic membrane (which is recalled to serve as a link in the transmission of acoustic energy from the ear canal toward the inner ear) should become less compliant due to loading (The latter can be expressed differently by saying that the resonant point of the air cushion moves toward higher frequencies) In other words, the occlusion phenomenon becomes less effective due to *attenuation both at the source and in transmission* This, in the opinion of

the present authors, is the explanation for the above-mentioned observation of Békésy that a deep-seated occlusion fails to produce the occlusion effect

Finally, when the occlusion is affected directly at the tympanic membrane (cf Fig 12) a bone conduction loss must ensue, not only because of the impairment of the ossicular system as stated in paper No I, but also because of the elimination of the external-canal contribution.

## 4 *Bone Conduction Contribution of the External Ear Canal*

The present findings gave evidence for a contribution to the total bone conduction response arising from the external ear canal In terms of the older terminology, this contribution belongs clearly to the "osseo-tympanic" mode of bone conduction of Bezold (1885). It consists of two components (1) Energy radiated into the external ear canal and transmitted from there *via* the middle ear, and (2) an air load upon the tympanic membrane similar to that constituted by the air enclosed in the middle ear, a bone conduction factor which was originally postulated by Groen (1962) and was shown to exist in paper No I of the present series] At present, there is no experimental way of separating the two components arising in the external canal from each other

In re-checking the "definitions" of bone conduction components in paper No I, the reader will realize that the combined contribution of the external ear canal had remained part of the component labelled "ossicular inertia" In the studies underlying papers No II and III, bone conduction responses had been obtained before and after amputation of the pinna and the external canal It stands to reason that the latter intervention minimized the bone conduction contribution of the external canal, since, especially in the cat with its very short osseous ear canal, the tympanic membrane then faces directly the outside

Fig 14 shows the relative magnitudes and phases of the bone conduction contribution of the external ear canal evaluated from the data of papers No II and III Given in the same figure is the *revised* ossicular-inertial component It must be mentioned that, in contrast to most other components, the between-animal variation with respect to the external-canal contribution was fairly large This fact may find its explanation by variations in size, shape, and detailed configuration of the pinna as well as the unavoidable variations in its position at the time the measurements were taken

Fig 14 suggests (although this cannot be stated with certainty from a graph depicting relative magnitudes) that the external-ear contribution to bone conduction is made up of a series of resonances (at 250 Hz, 1000 Hz, and 4000 Hz) and alternate anti-resonances (at 500 Hz, 2000 Hz, and 8000 Hz) a notion which is in line with the findings of Fig 11 and the [illegible] of R[illegible]ne (18[illegible]) and Huizing (1960) Similar observa- [illegible] in other animal species (e g guinea pig, cf. Fig 5 of paper No III

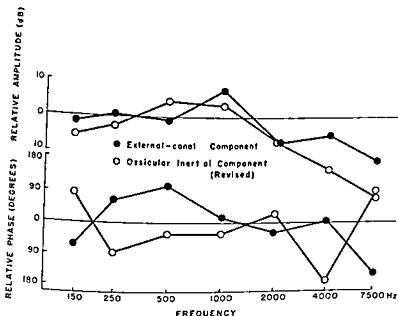



Fig 14 The relative contributions of (a) the external ear canal and (b) ossicular inertia (revised) to the total bone conduction responses amplitudes and phases for seven frequencies (calculated from results of papers I and III)

The large relative magnitude of the external canal contribution shown in Fig 14 explains why the cochlear microphonic responses had reflected the sound pressure readings taken in the external canal as closely as they actually did (cf Fig 6) If of lesser importance, its effect might have been completely obscured by that of other, more dominant, components

Comparison of Fig 14 with Fig 13 *b* of paper No I indicates that the splitting off of the external-canal contribution had made the curve representing the ossicular inertial component conform better to the classical notion, i e a large contribution in the low frequencies becoming less dominant in the higher frequencies It now presents itself as a curve, practically flat at low frequencies and after a small peak in the region of its own resonant point at 600 Hz, decreasing approximately with 9 dB octave That is to say its contribution to the total bone conduction response is largely limited to the frequencies *below* the resonant point of the ossicular system However, it is curious to note that contrary to the prevailing current opinion the ossicular component does not determine the total bone conduction response at the lowest frequencies Here, the external canal contribution is dominant

It has been postulated by some of the adherents of Bezold s concept of "osseo-tympanic bone conduction that *any* structure of the conductive mechanism might actually contribute to the total bone conduction response in essentially the same manner as was shown here for the external canal contribution For example a similar contribution might exist in the middle

ear, i.e. its walls might radiate acoustic energy into its lumen which would be picked up by the tympanic membrane. If this were so, opening and closing of the *bulla tympanica* should also reveal a high-pass filter effect. A re-check of pertinent data of paper No. I failed to give evidence for such a notion. Thus, with respect to the air within the tympanic cavity, no other effect than that due to the compliance load could be demonstrated.

## Appendix A. The Mechanisms Underlying the Clinical Tests of Bing, Rinne, Weber, and Gellé

Explanation for the above named clinical tests, based upon currently accepted Theories of Bone Conduction, are at variance in many instances with clinically observable facts as was mentioned in the introduction to the present paper. The studies reported here suggested several modifications of the general theory. Therefore, the present authors offer new (or modified) explanations for these tests, based mainly upon the results of papers No. I, II, III, IV, and VI. It is their considered opinion that these explanations will be in better agreement with clinical experience.

The tests of *Bing*, *Weber* and *Gellé* make use of a subjective phenomenon, the lateralization of a signal perceived by both ears and fused into one common image. Bone conduction signals, especially when applied to the forehead, reach the regions of both ears approximately with the same intensity. Lateralization of continuous tones, as is well known, may be caused by intensity and/or phase differences between the two ears, i.e. the fused sound image appears to be shifted toward the side of the ear in which either the intensity is higher or the signal phase is leading. According to Sedee (1957) phase advances in one ear can override small intensity differences of opposite tendency up to approximately 6 dB, i.e. lateralization in such cases will occur toward the ear with the leading phase.

(1) The test of *Bing* (1891) sometimes also referred to as the "physiological Weber test", rests upon the following phenomenon. Occlusion of one (normal hearing) ear produces lateralization of low-frequency bone-conduction signals to the side of the involved ear. A failure to lateralize indicates an impairment of middle-ear function.

The findings of the present paper (No. V) provide an explanation for this phenomenon. The occlusion effect involves the contribution of the external canal to the total bone conduction response. Occlusion eliminates a high-pass filter constituted by the external opening of the canal (or attenuates its effect), and this results into a relative low-frequency emphasis. Thus the lateralization phenomenon rests upon an *intensity difference* between the two ears. The effect requires a functioning middle ear for proper transmission of the improved response toward the inner ar. Hence it is abolished when middle ear function is impaired.

(2) In the test of [illegible] (1823), the ear canal is filled with water. This also produces [illegible] lateralization of low-frequency bone-conduction signals

toward the side of the involved ear The lateralization fails to materialize in fixation of the stapes footplate

The clue to this test was given in paper No II Fig 6 A mass loading of the tympanic membrane improves low frequency bone conduction responses due to an increase in the moment of inertia of the ossicular system Therefore the lateralization phenomenon is once more based upon *intensity differences* between the two ears Stapedial fixation eliminates the effect of the ossicular inertial bone conduction and thus of course its modification by tympanic membrane loading

(3) In the test of *Weber* (1834) various pathological conditions of the middle ear may produce lateralization of bone conducted signals (mainly in the low frequencies) toward the side of the involved ear Here the explanation depends upon the specific condition

(a) *In middle ear effusion* and *simple otitis media* the response to bone conducted signals at and below the resonant point of the middle ear improves slightly This finding is explained (cf paper No VI Fig 4) as a combined effect of mass loading of the drum membrane and increased friction (the latter being due to the fluid accumulation in the middle ear) Lateralization once more is due to an *intensity difference* between the two ears

(b) *In stapedial fixation* and *ossicular discontinuity* a different mechanism is operating Fig 5 *a* of paper No I had shown that both conditions are characterized by pronounced positive phase shifts in the frequency range at and below the resonant point of the middle ear for bone conduction At frequencies well below the resonant point where the concomitant response loss is small the phase shift will override the latter Hence lateralization is due to the *phase lead* in the involved ear In the region of the resonant point the response loss as a rule is too large and thus will not be overcome by the phase lead so that lateralization occurs toward the side of the uninvolved ear Clinically (S N Reger pers comm) lateralization in those frequencies (around 2000 Hz in man) frequently occurs toward the side of the uninvolved ear Observations in individual cases are often complicated by concomitant sensorineural losses in the higher frequencies

Groen (1962) had come to essentially the same conclusions (1) based upon the earlier observations of Legouix & Tarab (1959) concerning positive phase shifts of bone conduction responses upon impairment of middle ear function and (2) based upon the finding of Sedee (1957) i e that phase advances in one ear can override small intensity deficits of 5 6 dB In addition he had noted that in the region between 1000 Hz and 2000 Hz lateralization often changed abruptly from one side toward the other with small changes in frequency when the test was carried out with a sweep frequency signal Groen explained this finding with variations in the resonance of the middle ear cavity effect an explanation which the present results in the cat could *not* confirm (cf paper No I) It appears more

probable that such shifts *from* side *to side* within this *particular frequency* range are due to the competition between positive phase shifts (gradually decreasing with frequency) and response losses (gradually increasing with frequency up to the point *of middle*-*ear* resonance) in the involved ear, as was just outlined above

The notion that intra-aural phase differences might be involved in the mechanism underlying the Weber test is actually an old one Bruenings (1910), Krainz (1926), Langenbeck (1954) have already held *it on* theoretical grounds Naunton (1957), expressed scepticism about its importance

(4) In the test of *Gellé* (1885), once *more* advocated by Arnold *in* recent years (1964), an increase of air pressure in the ear canal reduces responses in the involved ear, i e leads to a lateralization toward the side of the opposite *ear* The test fails with *impairments of* middle-ear function, e g in stapedial fixation

The following explanation is proposed The increased air pressure displaces *and* thus stretches the *tympanic* membrane, putting it *under* tension This increased tension *impairs* the *bone*-conduction *contribution* of the external as well as of the middle ear Simple consideration will show that a *decrease* in air pressure in the external canal should have the same effect This was indeed observed by van Dishoek (1952), in one of his numerous experiments with the so called "pneumophone"

Again when there is a stapedial fixation, the transmission of the external-canal contribution as well as the response of the middle ear is already reduced or even *eliminated* so that tensing the tympanic membrane fails to produce changes in the bone-conduction responses

This enumeration has shown that the mechanisms underlying these four tests, the outcome of which is so similar (except that in the Gelle test lateralization occurs toward the opposite side), differs slightly but significantly from one another Bone conduction is too complex a phenomenon and defies explanations on the basis of a *single* factor

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# VI. THE RELATIVE EFFICIENCY OF AIR AND BONE CONDUCTION IN CATS

JUERGEN TONNDORF, ELLIOT C. GREENFIELD, AND ROGER S. KAUFMAN

## Summary

*In experiments in cats*, signals of identical frequency were applied simultaneously via air- and bone-conduction channels in such a way that the cochlear microphonic output was cancelled. (The bone input was located on the side of the head.) By definition, the responses to either signal must then be equal in magnitude. The two inputs, for air and for bone, were compared in terms of the applied forces and their phase relationships were determined.

With the exception of the low-frequency region, the vibratory forces required were larger, by several orders of magnitude, than the acoustic forces. This finding indicates that, in general, transmission is much more efficient via the air-conduction pathways than via those for bone conduction. It was only in the low-frequency region, below 600 Hz, that the acoustic forces approached the vibratory forces, especially when the middle-ear function was impaired. These findings have implications for the problem of hearing one's own voice.

The results of the phase measurements indicated that, when cancellation of the cochlear microphonics was achieved, only the cochlea itself was at rest. This finding gives once more evidence to the fact that the external, middle, and inner ears respond actively and independently to bone-conduction stimulation.

Lastly, the present results permitted to present the eight bone conduction components derived earlier in terms of their actual response curves.

## A. Introduction

Bekesy's cancellation experiments (1932) must be considered one of the key contributions to Bone Conduction Theory. The fact that a signal simultaneously transmitted to the ear via air- and bone-conduction channels

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can be brought to produce an auditory null for the observer (by adjusting the amplitude and phase relationships between the two channels) demonstrated clearly that the two modes of transmission must excite the cochlear receptors in essentially the same manner

Lowy (1943) performed one supplementary experiment He produced air/bone cancellation in laboratory animals by using cochlear microphonics as the response indicator He made the important additional observation that once cancellation was achieved for a given electrode position (e g at the round window) it was maintained for all other positions along the cochlea In other words the entire cochlear partition was found to be at rest

Bárány (1938) was able to show that for a single low frequency signal (435 Hz) the phase relationship between the air and bone conduction inputs varied with the location of the bone vibrator about the head Invariably the air conduction signal was delivered by an earphone attached to the ear The systematic variations Barany found provided good support for his concept of ossicular inertial bone conduction

Two questions still remained unanswered (1) What is the amplitude ratio and phase relationship between air and bone conduction inputs for a broad range of frequencies? (Bárány s experiments as was just mentioned had answered this question for one frequency only ) (2) Which parts of the peripheral organ really come to rest when cancellation of the cochlear output is achieved? (Lowy had tested this for the inner ear only )

## B Procedure

Cats were used in the present experiments The method of anesthesia and animal preparation was essentially the same as that employed in the earlier studies (cf papers I and X) Cochlear microphonics were once more used as the response indicator Round window electrodes were utilized since no middle ear alterations were intended An indifferent electrode was placed into the neck muscles

The air conduction signal was delivered by a loud speaker driver (Jensen DD 100A) coupled to the ear by a piece of rubber tubing 35 cm long and 1 2 cm in inside diameter in order to minimize drum membrane loading The rubber tube terminated in a small plastic insert which was tightly fitted into the external ear canal The air signals were monitored by a probe microphone (Bruel and Kjaer type 4134 microphone in conjunction with a probe 1 mm in diameter and 40 mm in length from the UA 0040 kit) The probe was inserted into the osseous ear canal according to a method of Pfalz (1969)

The bone conduction signals were delivered by a bone vibrator used previously in other such experiments (Western Electric D 80904) This instrument was solidly connected to an accelerometer (Bruel and Kjaer type 4308) which served to monitor the vibratory input The accelerometer in

tura was rigidly fastened to the skull bones at a place approximately 1.5 cm in front and slightly above the external ear canal.

The instrumental arrangement was essentially the same as that employed in paper No. V (cf. Fig. 1, loc. cit.). All voltages were read via the same $^1/_3$ octave band filter (Bruel and Kjaer, type 2112). Noise levels per pass band were in the order of 0.5 to 0.7 µV so that cochlear microphonic responses of 5 to 10 µV could be read routinely throughout the experiments (S/N = 20 dB). The method of obtaining phase readings (*re* the electrical input signal before power amplification) and their evaluation will be described in greater detail in Appendix B.

Cancellation of the *microphonic output* was achieved as follows. An air-conduction signal which produced a standard response of 5 µV was introduced. Then the *bone-conduction signal* was added and varied in amplitude and phase until the *response* was cancelled. Cancellation was always carried to the limits imposed by the noise level, i.e. as a rule the response was reduced by approximately 20 dB in amplitude. Finally, amplitudes and phases at the two *inputs* as well as the phases of the microphonic responses were assessed separately for air and bone inputs.

By definition when cancellation is achieved the air and bone conduction responses *must be precisely equal* in amplitude but in phase opposition. However, *in order to compare* input magnitudes in the two channels, a common denominator had to be established. The probe microphone read *sound pressure* (g · cm$^{-1}$ · sec$^{-2}$) and the accelerometer acceleration (cm · sec$^{-2}$). Both readings were converted into units of *force* (g · cm · sec$^{-2}$). This was achieved (a) by multiplying the microphone readings by the area of the tympanic membrane and (b) by multiplying the accelerometer readings by the *mass* of the animal's head. According to Wever and Lawrence (1954) the area of the tympanic membrane of the cat is 0.4 cm (= −8 dB). The latter value was employed *routinely*. The weight of the head was determined after termination *of each experiment*, i.e. after the sacrifice of the animal. It was found to vary *between* 170 g and 230 g for cats ranging from 1.8 kg to 3.2 kg in body weight. Thus if one considers 200 g a representative value the mass would be 0.2 g (= −14 dB). Individual corrections were made.

Several animals served to establish the procedure. Amplitude data were obtained in a series of six *animals*, complete phase data in only three of them. Three animals with subacute otitis media simplex were encountered accidentally. Amplitude measurements were taken in all of these *animals*, complete phase data in only one of *them*.

Barany (1938), it is recalled, *used* a single test frequency but *varied* the location of the bone vibrator about the head. His *results* were *presented* in the form of *amplitude/phase* vectors with the air input as the *reference*. By contrast, in the present *experiments* the location *of the* vibrator was kept constant at the side of *the* head but frequency was varied. Eighteen frequencies *between 100* Hz and 7500 Hz were *routinely* employed.

## C Results

Fig 1 shows an example of the amplitude ratio between the magnitudes of the bone and air inputs (in terms of force) as well as the phase differences between the same points in a single animal with a normal middle ear, after cancellation had been completed Because of the large variations with frequency of response amplitudes (or their ratios as seen in Fig 1) results cannot be presented in vectorial form but will be considered separately in terms of amplitudes and phases

### 1 *Amplitudes*

(a) *Normal middle ear*—Fig 2 gives the amplitudes (in terms of forces measured at the air and bone inputs) required for cancellation once more for a single animal It is noticed that whereas the air input curve follows a fairly smooth course the bone input curve appears more irregular The same irregularity was reflected in the ratio curve of Fig 1 (It is recalled that both curves for air and bone were obtained for a constant cochlear microphonic output ) Paper No IV (Fig 4) had provided an explanation for the irregular appearance of the bone input curve It had been found in cadaver experiments on cats that the transmission of vibratory energy from an input at the side of the skull to the lateral vestibular wall varied irregularly with frequency up to and sometimes in excess of ±10 dB The

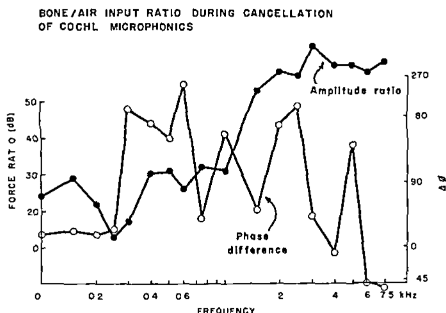



Fig 1 Ampl tude ratios (in terms of force) between the bone and air inputs to a cat s r dur ng cancellation of the cochlear microphonic output (round window electrode) F s relat onsh ps for 18 frequencies between 1000 Hz and 7500 Hz data from an mal

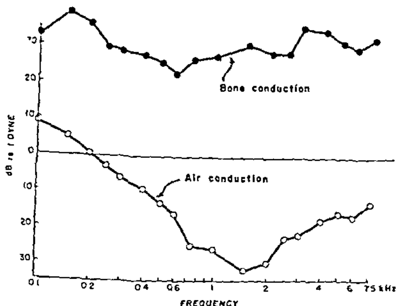



Fig 2 Force at the air and bone inputs (sound pressure × drum membrane area and acceleration × mass of animal s head) during cancellation of the cochlear microphonic output (round window electrode) data from one single animal

manner in which it varied was practically unpredictable, except that the support of the animals head seemed to be a major contributing factor

That the variations of the bone input curve were essentially random is shown by the fact that by averaging the results of a number of animals (even over the small number of six animals) the bone input curve became much smoother (Fig 3) The points of maximal sensitivity were 2000 Hz

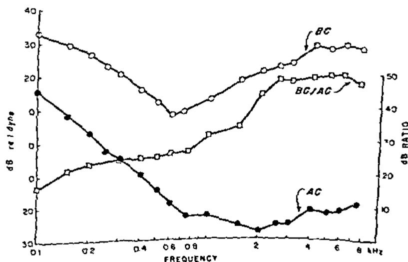



Fig 3 Same as in Fig 2 for an average of six normal animals The ratio between the two curves (BC AC) is also given

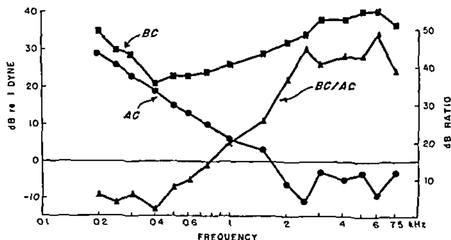



Fig 4 Same as in Figs 2 and 3, average data for three animals with pathological middle ears

and 600 Hz for air and bone inputs respectively, confirming once more the disparity of the resonance points as seen by air and bone inputs (cf paper No III) In addition to the forces at the air and bone inputs, Fig 3 gives their ratio as a third curve Inspection of the three curves indicates that, in general, the force required at the bone input is higher than that at the air input, a fact attesting to the efficiency of the middle ear as an impedance matching device It is only in the low frequencies that the two input curves even came close to each other At first, both curves ran almost parallel, the ratio curve having a slope of only 3 dB/octave Beyond the resonant point of the bone-conduction curve (at 600 Hz), the ratio increased very rapidly with frequency (approximately at 12 dB/octave), to level off at a constant value of about 50 dB beyond the resonance point of the air input curve (at 2000 Hz)

(b) *Pathological middle ears*—The present series, as was already mentioned, included three animals with subacutely infected middle ears That is, on inspection, the tympanic membranes had been found closed, somewhat thickened, but not bulging However, the bulla and middle ear when opened were seen to be filled with muco-purulent material, the mucous membrane being greatly thickened Landmarks were defined with great difficulty Fig 4 gives the averages of the results obtained in this group A comparison with the normal animals of Fig 3 shows that the air-conduction sensitivity was reduced, especially in the low frequencies The bone input curve was somewhat flatter and its resonance point was shifted toward lower frequencies (from 600 Hz to 400 Hz) In the region below the resonance-point for bone, the air and bone input curves ran much closer t[illegible]ther than in normal animals Beyond this point, the bone/air ratio cur[illegible]e much more steeply with frequency than in normal animals, i e with a s[illegible] (compared to 12 dB/octave in normal animals)

to level off approximately at 45 dB beyond the resonance point of the air input curve (2500 Hz).

2 *Phase*

Fig 5 gives the phase angle between the bone and air inputs (accelerometer and probe microphone respectively) during cancellation of the cochlear microphonic responses Results are given for three animals with normal middle ears (The animal from Fig 1 is included ) The agreement between the three curves is fairly good, at least with respect to their general shape

In general, there is a tendency for the bone input to lead the air input, with several peaks clearly discernible at 300 Hz, 600 Hz, 1000 Hz, 2500–3000 Hz, and 5000 Hz The average phase difference at these five points is reasonably close to 180°. It is interesting to note that in Barány's experiments (1938), which were conducted in human subjects and confined to a single frequency of 435 Hz, the bone input also lead that for air by approximately 180° when the vibrator was applied to the side of the head However, the notion that the phase opposition between bone and air inputs for this particular vibrator placement might exist for *all* frequencies is an over simplification The phase relationship as Fig 5 indicates, is not independent of frequency

Figs 6 and 7 give the phase relationships between the microphonic responses to air and bone conducted signals with reference to their respective inputs

When responding to *air borne* signals (Fig 6), the phases of the cochlear microphonics at low frequencies, up to approximately 500 Hz, coincided with those of the signal Thereafter, the phase relationship rapidly ap

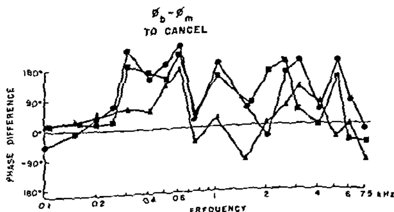
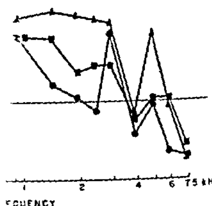



Fig 5 Phase differences between bone and air inputs ($\Phi_b$, $\Phi_m$) during cancellation of the cochlear microphonic output (round window electrode) Data from three normal animals (Note that some phase differences are given >180 in order to demonstrate average trends )




... hlear microphonic outputs and the air inputs ... shown in Fig 5

d 180 with the cochlear microphonic response leading At higher f... its phases changed more rapidly with frequency and individual ... became more pronounced This finding confirms the well established fact that at low frequencies the entire middle-ear system transmits like a rigid piston.

When responding to *bone conducted* signals (Fig 7), the phases of the cochlear microphonics at low frequencies lead those of the signal input by approximately 180° Once more, at higher frequencies, the relationship changed rapidly with frequency, although the agreement between animals was quite good Since at low frequencies the ossicular inertial component is rather dominant (cf paper No V, Fig 14), it is fair to state that, according to Fig 7 of the present paper, the response of the ossicular system at these frequencies is in phase opposition to the input, provided the latter is from the side of the skull, but, again, this simple relationship prevailed

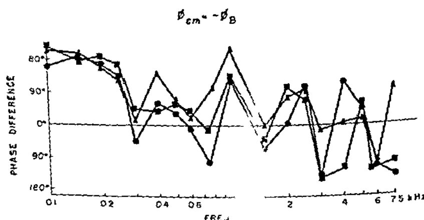



Fig 7 Phase differences between the cochlea[r m]icrophonic outputs and the bone inputs ($\Phi_{cm} - \Phi_b$) for the same three animals show[n i]n Figs 5 and 6

only at frequencies below the resonant point of the middle ear for bone-conduction (600 Hz). In view of the mode of bone conduction response of the ossicular system (cf. Bárány, 1938, paper No. I), this is a logical finding.

## D. Discussion

The response curves in terms of the force applied *via* air- and bone-conduction (Fig. 3) call for some comments with respect to the problem of *hearing one's own voice*. Bekésy (1949), in a related study of his own, pointed out that a speaker's voice is carried to his ears both by air- and by bone-conduction. He was able to show that in the case of man there is a considerable attenuation along the air pathway due to the directionality of the output from the mouth: approximately 20 to 25 dB from the lips to the entrance of the ear canal. If, for the case of the cat, the attenuation is assumed to be of the same order of magnitude, the forces generated by its voice and acting upon the ear simultaneously *via* air and bone channels may be approximately equal to each other at frequencies below 600 Hz. At higher frequencies, however, the transmission by air will always be at a clear advantage.

This situation cannot be too far from that prevailing in man. It accounts for the fact (as Békésy pointed out already) that everyone when hearing his own voice from a recording for the first time is struck by its apparent "tinniness", i.e. by the lack of bass. The bass emphasis one is accustomed to when hearing his own voice in a normal fashion is of course due to the bone conduction contribution which is absent from the recording. With a *middle ear transmission loss* (Fig. 4), the bone pathway may well be superior in the low frequencies, accounting for the well known "boominess" of one's own voice when suffering from an otitic catarrhal infection, for example.

Fig. 8 gives the ratios between the air and bone responses of Fig. 4 (pathological ears) and those of Fig. 3 (normal ears), plotted in the manner of an audiogram. The recordings of Fig. 4 were taken with the active electrode placed upon the inflamed and greatly thickened soft tissues in the vicinity of the round window. Some electric attenuation was bound to occur. Indeed a sharp electrode, introduced through the soft tissues into *scala tympani* gave output readings which were increased approximately by a factor of three. Furthermore, it had been found earlier that middle ear impairment did not affect the bone conduction responses to higher frequencies (cf. Paper No. I). The zero line of Fig. 8 has therefore been corrected to the 11 dB level, the average of bone-conduction responses in the higher frequencies.

Taking this correction into account (dashed line of Fig. 8), one sees that air-conduction responses have suffered a flat loss of approximately 20 dB in the low frequencies. This loss diminishes gradually with frequency

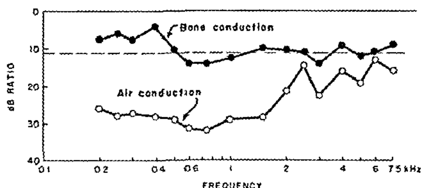



FIG 8 Amplitude ratios (in dB) between the data for air and bone of Fig 3 (normal animals) and Fig 4 (pathological animals), drawn in the manner of an audiogram. Note the correction of the zero line, necessitated by the electric attenuation between the cochlea and the electrode in pathological ears (for details cf text)

starting approximately beyond 1500 Hz The bone-conduction curve shows only a slight loss around its normal resonant point (600 Hz), but a small *improvement* at lower frequencies This latter observation is in agreement with clinical experience (Lierle & Reger, 1946, Rytzner, 1954)

For an explanation one must compare once more Figs 3 and 4 These figures indicated that in pathological middle ears the bone-conduction curve is somewhat flatter than that obtained in normal ears, and also that the resonance point has shifted downward from 600 Hz to 400 Hz These two facts lend themselves to an explanation of the underlying mechanism The downward shift of the resonance point, according to the findings of paper No II (Fig 6), suggests a *mass loading* of the middle-ear system, probably due to the thickened and inflamed soft tissues The flattening of the curve is compatible with the assumption of an *increase in damping* probably due to the accumulation of fluids in the middle ear That the latter effect did not cause an overall reduction of the response (note the corrected zero level in Fig 8) is accounted for by the fact that a mass load of the ossicular system increases the latter's *moment of inertia*, and this apparently makes up for the loss due to damping

*Phases* as is recalled, were measured at both inputs (air and bone) and at the round window electrode, independently for both driving modes, after cancellation had been achieved All phase measurements were referred to the electrical input signal going into the power amplifier (For further details of phase computations cf Appendix B ) Inspection of Figs. 5 to 7 indicates that when cancellation was achieved at the round-window electrode [and thus, according to Lowy (1943), along the entire cochlea], sound pressures in front of the tympanic membrane had not been canceled It appears, therefore in answer to the question raised above, that the [illegible]n *of the cochlear output entails only an arrest of movement* [illegible]n *the cochlea itself* This impression was double-checked Cancella[illegible] was carried out for the probe microphone in front of the tympanic

membrane In that case, the cochlear microphonic output from the round window was reduced, but clearly present Under those circumstances, the air input and the bone contributions of the external and middle ear (cf paper No V) must have canceled one another, and the middle ear must have been at rest Thus *the cochlear response obtained must have been due to the bone conduction response of the inner ear alone with the external and middle ear intact, but functionally eliminated* In paper No I the same effect had been achieved by *amputation* of the external and middle ear structures and/or mechanical fixation of the tympanic membrane (Fig 5, loc cit ) It ought to be mentioned that the response curve obtained upon cancellation at the probe microphone in the present experiment was very similar to that upon mechanical fixation of the tympanic membrane of paper No I These findings are once more evidence for the fact that the external canal the ossicular chain, and the inner ear respond *actively and independently* to vibratory stimulation, although it does not give any direct information about the specific mode of the inner ear response (By inference, this response can hardly be anything else but compressional For more direct evidence in this latter respect cf papers I and IV )

## E Absolute Response Curves of Bone Conduction Components

Since care was taken that all recordings were obtained well within the linear portions of the cochlear input output curves, the bone-conduction curve of Fig 3 can be readily converted into a response curve for constant input The availability of such a curve permits re evaluation of the various bone conduction components assessed earlier (papers I & V) but now in the form of actual response curves Earlier the components were given merely relative to the total bone conduction responses a method which did not permit any statements as to *resonance points anti resonances, etc* In the following five figures (Figs 9 a c, 10 and 11) the components evaluated so far and some of their combinations are re drawn for a constant input level at the side of the animal's head In every figure the total bone conduction response curve is included for convenient comparison

Fig 9 a gives the bone conduction contributions of the external and middle ear That of the *external ear canal* represents (a) the transmitted portion of the *sound radiated into the external canal by its own walls* and (b) the *load of the air column within the canal upon the drum membrane* Experimentally these two components could not be separated The total contribution is given by a curve with a single resonant point at 1000 Hz and relatively steep slopes on both sides The other resonant points suggested by the relative response curve (cf paper No V Fig 14) are no longer discernible

The two *middle ear components* are also shown in Fig 9 a The *ossicular inertial component* has a single peaked response curve with relatively

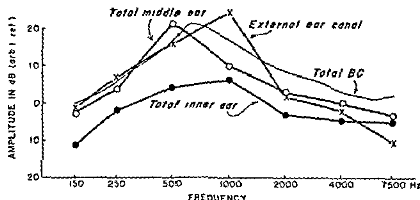



Fig. 11. Frequency response curves of the bone-conduction contributions of the external, middle and inner ear.

should become more effective as frequency goes higher. This prediction, as is seen in Fig. 10 is really borne out only for the "pure compressional component" with its sigmoid shaped curve, although the difference between low and high frequencies is not very great. Actually, in terms of the relative response curve (cf. paper No. I, Fig. 13 *b*), the difference between low and high frequencies is somewhat larger, but, then again, this is only so because the overall response curve slopes off toward higher frequencies, and it has nothing to do with the response of the inner-ear *per se*. When other inner-ear components are added (either the two window releases or all of them) the curve shape, as Fig. 10 shows, alters completely. In both of the latter two cases there is an emphasis of the middle frequencies between 500 Hz and 1000 Hz.

Fig. 11, finally compares the lumped contributions of the external, middle, and inner ear respectively. Actually, all three curves peak in the middle frequencies. It seems that the point of maximal sensitivity of the overall curve is mainly determined by the contributions of the middle and external ear. However, it was mentioned earlier that it coincides with the resonant point of the ossicular system. In fact, it did not alter appreciably either with the amputation of the pinna and external canal (elimination of the canal contribution) or with the opening of the bulla (elimination of the middle ear compliance effect).

Needless to say, the present description applies only to the frequencies at which measurements had actually been carried out. Additional peaks or dips may have been missed at intermediate frequencies and/or outside the range covered. With respect to intermediate frequencies, the likelihood of this occurrence is not too great since some of the earlier measurements of paper No. I had been repeated with the denser frequency spacing of the present instrumentation. With respect to frequencies outside of the present range there is one additional datum. In a different set of experiments (Kaufman, Tonndorf, and Khanna in press), bone conduction

measurements were carried out in cats at a frequency of 14 500 Hz with the aid of a tuned bone conduction vibrator At this frequency the amputation of the middle ear scarcely affected the bone conduction responses This finding shows that at this high frequency the inner ear has actually become the dominant factor, a trend which is indicated in Fig 9 c

Corso (Corso, 1963 Corso & Levine 1963) has recently revived the old notion of extra cochlear i e of direct neural reception of bone conducted sounds He based his hypothesis upon an exploration of high to ultra high frequencies by bone and air-conduction He found that in contrast to lower frequencies, the loudness and pitch function at these frequencies are different for bone and air conduction

The present experiments, of course were not designed to test such a proposition However one remark may be made in regard to this question In the experiments of Kaufman et al (in press) just mentioned the microphonic response to bone conduction was explored up to 20 000 Hz The response curve was continuous and did not show any breaks indicating a termination of cochlear responses in that frequency range

In view of the complexity of the response of the ear to bone-conducted sounds (cf Figs 9 through 11), which is contrasted to that by air conducted sounds the present authors hold it quite possible that the various contributory modes and components have different limits of linearity This could well affect both loudness and pitch judgements especially if subharmonics were generated by one or another of the receptor modes

Be that as it may it is quite conceivable that in contrast to air conduction which is limited by middle ear transmission compressional bone conduction remains effective to rather high frequencies This might account for some of Corso s findings

## Appendix A The Traveling Wave Patterns Along the Cochlear Partition in Response to Compressional Bone Conduction

The present results have confirmed the earlier results of Békésy (1932) and of Lowy (1943) i e that the cancellation between air and bone-conduction signals is a purely peripheral phenomenon Specifically it is now clear that this cancellation takes place in the inner ear alone This latter finding necessitates the conclusion that the cochlear response pattern to air borne and to vibratory stimulation must be precisely identical Békésy (1928) first determined the response pattern of the cochlea to air borne sound the well known *travelling wave pattern*

The difficulty in equating the air and bone responses of the cochlea lies in the fact that on the one hand i e for air conduction and the bone contributions of the external and middle ear the energy is transmitted to the cochlea at *one* point the stapes and *waves travel away from their source* on the other hand i e for compressional bone conduction the

input must be assumed to be diffusely distributed over the entire cochlear shell How the *distortional vibrations* of the cochlear shell (which are the essence of compressional bone conduction) produce a displacement of the basilar membrane, has been explained in paper No I However, this latter explanation did not account for the generation of traveling waves or for the direction such wave travel may take Békesy (1942, 1955) was able to show, with respect to the second of the above problems, that regardless of the location of the cochlear input, waves invariably travel from the stiffer portion of the cochlear partition toward the more compliant one, i e from the base toward the apex In the extreme case, when the input was located directly at the apex, the waves traveled *paradoxically against their own source*

This peculiar and invariable response pattern of the cochlear partition can be more fully explained in the following manner [The following account has first been presented by one of the authors following his own studies on cochlear models (Tonndorf, 1962) It must be noted, however, that it agrees completely with the conclusions reached *earlier* by Zwislocki (1953) on the basis of his mathematical considerations The senior author wishes to take this opportunity to apologize to Dr Zwislocki for his oversight at the occasion of the original publication (1962) ]

Consider once more the extreme situation referred to above Suppose a high frequency signal is made to enter *scala vestibuli* in the apical region by any means whatsoever This signal causes a fluid displacement which will propagate mainly in the direction of the least impedance, i e eventually toward the round window [According to Kirikae (1960), the round window is approximately 20 times more compliant than the oval window ] In the pathway of this fluid motion lies the cochlear partition which, consequently, must be displaced also The cochlear partition can be represented, in essence, by a series of low-pass filters which are arranged in such a way that their cut-off frequencies vary systematically from apex to base (This *filter property is a direct corollary of the stiffness gradient mentioned* above ) It is clear, then, that the cochlear partition cannot be primarily displaced in the apical region when the signal frequency is high, as was assumed in the present example The stimulus must first find a "gate", i e a region along the partition where the pass band is wide enough to include the signal frequency For a high frequency, this region must be near the cochlear base When eventually displaced here, the partition acquires potential energy which it releases in the form of a traveling wave, the latter progressing along the partition once more in the direction of least impedance i e toward the cochlear apex (This impedance gradient is another corollary of its stiffness gradient )

The mechanism just described implies that for any types of input, near [illegible] r near the apex narrow or broad, the cochlear response pattern must be precisely the same that is traveling waves are established, and they progress invariably against the cochlear apex

## Appendix B Evaluation of Phase Differences

First, a few definitions must be introduced Phase angles, as *measured* in reference to the electric signals in front of the power amplifier, will be labeled $\vartheta$ Those calculated, e g between the cochlear microphonics and the probe microphone, etc, i e relative to a reference other than the standard, will be labeled $\varphi$ To designate points of reference or measurement the subscripts $_r$(std reference), $_v$(variable phase), $_c$(phase angle *re* standard, measured during cancellation), $_m$(probe microphone), $_b$(bone monitoring accelerometer), $_{cm}$ and $_{cm}$ (cochlear microphonic responses to air and bone respectively), will be employed

(1) Suppose the phase angle at the output of X in Fig 12 was under consideration X may be the probe microphone, the bone monitor, or the cochlear response for either air or bone The phase shifter was varied until the difference as displayed on the oscilloscope became 0° $\vartheta_v$ is the phase angle measured in order to fulfill the proposition

$$\vartheta_v + \varphi_x - \vartheta_r = 0^\circ. \tag{1}$$

It follows from eq (1) that

$$\varphi_x = \vartheta_r - \vartheta_v \tag{2}$$

(2) During the cancellation experiments, the following circuit was used (Fig 13) Under the condition of bone air cancellation of the cochlear output, the phase angle $\varphi_c = \vartheta_v - \vartheta_r$ (or, since $\vartheta_r = 0^\circ$, $\varphi_c = \vartheta_v$) was obtained The phase

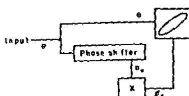



Fig 12 Block diagram of the circuit used for phase measurements (for explanations cf text)

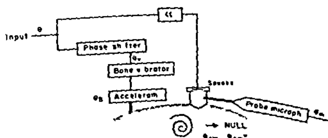



Fig 13 Block diagram of the circuit for phase measurements during cancellation of the cochlear microphonic output (round window electrode) (For explanations cf text)

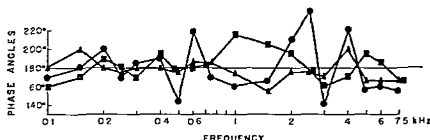



Fig 14 Errors in accumulated phase measurements along the air and bone channels including those of the cochlear outputs Ideally, the sum of accumulated phases in both channels ($\Sigma\Phi_{AC}+\Sigma\Phi_{BC}$), by definition, should be 180°

angles (corrected for the phase shift of the probe microphone), $\varphi_0$, $\varphi_{cm}$, and $\varphi_{cm}$ were then obtained in separate measurements with the aid of the general arrangement shown in Fig 12 Each of these phase angles was referred to the electric input [which happened to be the variable output of the phase shifter ($\vartheta_e$) going into the amplifiers of the bone and air conduction circuits respectively]

Of interest, among others, was the phase difference

$$\Delta\varphi=\varphi_b-\varphi_m \tag{3}$$

while cancellation was achieved This value could then be calculated (cf Fig 5)

In both channels, air and bone, several phases had been measured up to and including $\varphi_{cm}$ and $\varphi_{cm}$ Thus, the proposition,

$$\sum\varphi_{BC}+\sum\varphi_{AC}=180°, \tag{4}$$

provided the possibility of checking the accuracy of the various calculations leading up to eq (3) (By definition, the cumulative phase angles in both channels under the condition of cancellation of the cochlear output must be 180° ) If the result of

$$\varphi_{cm''}-\varphi_b=\Delta\varphi'' \tag{5}$$

and of

$$\varphi_{cm'}-\varphi_m=\Delta\varphi', \tag{6}$$

eq (4) after some rearrangement, may be re-written

$$\Delta\varphi-\Delta\varphi'+\Delta\varphi''=180° \tag{7}$$

Calculating $\Delta\varphi$ and $\Delta\varphi''$ and combining the results with $\Delta\varphi$ in the sense of eq (7) provided the desired check on the accuracy of the entire set of phase measurements involved in the cancellation experiments The results are given in Fig 14 for three animals with normal middle ears

on which complete phase data had been obtained Fig 14 indicates that an error value of $\pm 20°$ was not too frequently exceeded (somewhat more often at higher frequencies than at the low ones) This value is not too large considering the fact that underlying this calculation were four independent phase measurements at each frequency

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# VII. THE EFFECT OF OSSEOUS DISCONTINUITIES UPON THE TRANSMISSION OF VIBRATORY ENERGY ACROSS THE SKULL IN RATS

JUERGEN TONNDORF, MERLE OLESEN, ALOYSIA F. KING, ROBERT D COTTLE, AND DANIEL C BAKER III

### Summary

In rats, as in some other species (e g whales, bats), the temporal bone is not an integral part of the base of the skull but is connected to it by loose fibrous tissue Earlier writers had suggested that this anatomical feature might provide a structural isolation to minimize the transmission of vibratory energy toward the ear This notion was put to an experimental test The cochlear microphonics of rats in response to vibratory stimulation were measured before and after fixation of the temporal bone The effect was surprisingly small indicating that the protection afforded by this anatomical feature is not important

The fact that there were gains at some frequencies and losses at some others was taken to indicate that the changes observed upon fixation of the temporal bone might be due to an alteration in interference between competing pathways of transmission of vibratory energy across the skull This conclusion gave support to earlier findings concerning the transmission of vibratory energy through the interior of the skull in the form of pressure and/or translational waves This latter mode appears to occur in addition to the generally accepted form of transversal waves traveling along the bones of the skull

## A Introduction

When Barany (1938) established his concept of ossicular inertial bone conduction, he pointed out that its efficiency is fairly low because of the small moment of inertia of the ossicular chain This fact, he suggested, should protect the ear against undue reception of low-frequency vibrations which otherwise might interfere with its more important function the reception of air-borne signals The need for such protection has been demonstrated to anybody who ever experienced a bilateral middle-ear *effusion* D[illegible] t the Weber effect, one's own steps become disturbingly loud w[illegible] the same time the perception of air-borne signals is attenuated

[illegible] was mad[illegible] through the support by the following grants Research Otolary[illegible] [illegible]ning grant 5 TI NB 5234, Career grant 5K6 NB 21 691

Reysenbach de Haan (1957) had advanced an interesting hypothesis, based upon similar considerations, concerning the protection of the whale's ear against the reception of structural vibrations In whales, as he was able to confirm, the petro-tympanic bone is not an integral part of the base of the skull, i e its connections to the surrounding bones (the squamosal, the parietal, and the various bones making up the occipital) is by rather loose fibrous tissues Moreover, the petro-mastoid, which houses the inner ear, is almost entirely surrounded by a large number of air filled sinuses Reysenbach de Haan argued that the whale might well need such a protection as its tissues are much better matched in impedance to the surrounding water than is the case with land living mammals

Essentially the same situation, at least with respect to the mode of suspension of the entire tympanic bone, is known to exist in bats and in rats This fact has led other authors to considerations similar to those of Reysenbach de Haan with regard to the attenuation of bone conducted vibration [e g Henson (1961) concerning bats]

The rat has been used in previous experiments of the present series (papers No II and III) The tympanic bone, when exposed surgically in this animal, can be moved to quite some extent in reference to the parietal and occipital bones by application of relatively small forces In the earlier experiments just mentioned, the temporal bone was fixed routinely to the base of the skull by means of dental cement, a procedure which had resulted in some improvement of the bone conduction responses Nevertheless, the cochlear microphonic output of rats, which was used as the response indicator, is not very large as seen by a round window electrode Because of this a systematic investigation was impossible at that time Since then, a lock-in amplifier has become available which makes it possible to read responses routinely down to the 0 1 µv level

It was decided, therefore, to assess the bone conduction responses in the rat after fixation of the temporal bone to the base of the skull as compared to those in its normal unfixed state

## B Procedure

Three different strains of white rats (Wistar, Sprague-Dawley, and Sherman) were examined In all three (at least in young animals not fully grown) the temporal bone was found to be quite loose with respect to its surrounding bones There was no distinct difference among the three strains in this regard However, among all three of them, the incidence of subacute otitis media simplex was quite high In one instance, there were only three animals without infection in a batch of ten Prophylactic treatment with bicillin did not have a noticeable effect Finally pathogen-free animals (Manor Farm Statsburg N Y ) were obtained From then on otitis media was not encountered and the study was rapidly brought to a close

Young animals, weighing 220 g, as compared to an adult weight of 275 g, were used Anesthesia was by chlorpromazine, i e, 20 mg/kg, which was followed, 15 minutes later by urethane, intra-peritoneally, 0 7 g/kg The temporal bone and bulla area were exposed; the bones bordering on the temporo-parietal and the temporo occipital fissures were carefully freed of soft tissues to be ready for the later application of dental cement The styloid process in this animal incidentally, is part of the occipital The bones were kept moist to avoid drying up and subsequent hardening of the fibrous tissues A round-window electrode (100 μ silver wire with a small ball at its active end) was inserted and the bulla resealed An indifferent electrode was attached to the neck muscles The entire animal was placed on a foam-rubber cushion on its side The head, with the test ear facing upward, was held in place by long sutures through the rims of the surgical wound The bone-conduction vibrator (Western Electric D 80904) was supported from overhead in order to reduce its weight and to improve its frequency response Its output rod was fastened to the parietal bone above and in front of the external ear canal by means of dental cement An accelerometer (Bruel and Kjaer, type 4308), which had been made an integral part of the driving system, served to monitor the bone input Cochlear microphonics, in response to bone conduction signals were measured at the 0 1 μv level with the aid of a lock-in amplifier (EMC, type RJB) as already mentioned At this low level, all responses were within the linear portion of the input-output function In preliminary experiments, it was found that the spreading of the wound, necessary for the wide exposure of the temporal bone, practically closed off the external ear canal giving rise to an occlusion effect which was then masking other changes Therefore, a short piece of polyethylene tubing was inserted into the ear canal in order to keep it open

Input values for standard microphonic outputs (0 1 μv) were measured (1) with the ear normal (ear canal open), (2) after fixation of the temporal bone by dental cement, (3) after amputation of the middle ear (temporal bone still fixed), and (4) after freeing the temporal bone once more (In preliminary experiments conducted in a few animals, the temporal bone with the ear intact had been fixed and unfixed several times to assure that the original situation could be restored ) Step 4 provided a good check on the quality of fixation Grip cement (Caulk & Co ) was used throughout This material, when properly applied bonds strongly to bone Final evaluation of test results in this series was from eight animals which were run in succession once the procedure had been established in preliminary trials

Another series of six animals was used for the same tests, but with the vibrator placed on the forehead between the eyes In this latter procedure, the animal's head was kept in its normal upright position in order to ac[illegible] a perpendicular incidence of the vertically supported bone conduction vibrator In a few animals, an additional test was performed

in that the vibrator was shifted from its original position on the parietal bone onto the temporal bone which was left unfixed

## C Results

Fig 1 shows the changes in cochlear microphonics in eight animals for the following condition temporal bone fixed *vs* normal ear (ear canal open) The average trend is easily recognized When the ear canal was not kept open by an insert as described above the changes due to temporal bone fixation were less clear and the between animal variation was much larger because of the occlusion effect which was then very dominant

Fig 2 gives average cochlear microphonic changes (a) with the ear intact (ear canal open) and (b) after removal of the middle ear Both curves showed the same trends In fact in the high frequencies they were practically identical *In the low frequencies however there was a distinct* response change with the ear intact (cf Fig 1 for statistical distribution) but no change after destruction of the middle ear Even with the ear intact the response improvements were surprisingly small with the exception of the sharp peak (+14 dB) at 2000 Hz In the low frequencies improvements amounted to a few dB only while there was a slight but distinct response loss at 4000 Hz These observations indicate that the factor or factors responsible affects the bone conduction responses of the middle ear as well as those of the inner ear

When the bone conduction vibrator was removed from the parietal bone and placed directly onto the temporal bone without fixation of the latter the response alterations were essentially similar to those seen after temporal bone fixation with the driver remaining attached to the parietal bone The results shown in Fig 3 are from a single animal only The between animal variation was greater than in the first series a fact which might be largely attributed to the separation of the bone vibrator from and

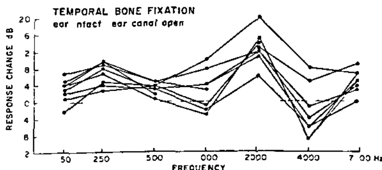



Fig 1 Response changes for seven frequencies between 150 Hz and 7500 Hz due to the fixation of the temporal bone to the base of the skull ear intact ear canal open bone conduction vibrator on the parietal bone data for eight animals

potential *entrance* for such energy to the cochlea There is really no contradiction in these two statements It is merely a matter of phase relationships between the events occurring at both ends of the aqueduct

The present results have indicated that the protection of the ear against structural vibrations in the range of audio frequencies afforded by the loose connection of the temporal bone to the base of the skull, as found in rats is really small The question whether or to what degree the pneumatized spaces surrounding the whale's labyrinth provide a structural isolation of the whale's labyrinth as also suggested by Reysenbach de Haan is presently open to conjecture Experimental data are not available in this respect

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S U P P L E M E N T U M 214

# CLINICAL PATHOLOGICAL CORRELATIONS IN SQUIRREL MONKEYS AFTER SUPPRESSION OF SEMICIRCULAR CANAL FUNCTION BY STREPTOMYCIN SULFATE

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and ASHTON GRAYBIEL

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SUPPLEMENTUM 214

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*Opinions or conclusions contained in this report are those of the authors and do not necessarily reflect the views or endorsement of the U S Navy Department*

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A systematic comparison between the responses of normal subjects and persons with bilateral loss of labyrinthine function exposed to unusual force environments has demonstrated that the vestibular end organs are essential to the causation of typical symptoms of motion sickness (Graybiel *et al* 1960, Meek *et al*, 1962, Johnson *et al*, 1962, Graybiel & Johnson, 1963) Even a partial loss of the vestibular function affords some protection, and among those so protected are patients who have received streptomycin sulfate for the treatment of Ménière's disease Schuknecht (1957) administered streptomycin sulfate to eight patients with Ménière's disease in amounts just sufficient to result in unsteadiness According to his investigation, no one suffered a loss in hearing, suggesting a selective affinity for the vestibular system Ten years later, four of his patients were reexamined, none experienced symptoms of motion sickness in the Slow Rotation Room and their hearing, when compared with the level before the treatment had improved in three and was unchanged in one (Graybiel *et al*, 1965 *a*) These findings suggested the possibility that in man streptomycin sulfate might suppress the vestibular function in normal ears sufficiently to prevent motion sickness and preserve hearing with a good margin of safety Moreover, in none of the reports describing the use of this drug in the treatment of Ménière's disease (Fowler, 1948, Hamberger *et al* 1949, Ruedi, 1951, Hanson, 1951) was there any mention of severe impairment of hearing

On the other hand, most pathological studies of the ototoxic effect of streptomycin sulfate in lower animals (Hawkins, 1947, Hawkins *et al*., 1950, 1956 Hawkins & Lurie, 1952, Winston *et al*, 1948 1949, Winston 1953 Causse, 1949, Ruedi *et al* 1949, 1952, Ruedi, 1951; Christensen 1951, Berg 1951, Lurie, 1955, Schuknecht, 1957; McGee & Olszewski, 1962, and others) had revealed some hair cell loss in the cochlea as well as in the cristae of the semicircular canals in normal ears Inasmuch as special differences were revealed, it seemed worthwhile to extend the studies to animals higher in the phylogenetic series, the better for extrapolation of the findings to man What follows describes our results using squirrel monkeys, and it may be stated here that the implications to be drawn from the clinicopathological correlations reach far beyond their significance for motion sickness

## METHOD

Eight healthy squirrel monkeys (Saimiri sciureus) were selected on the basis of normal semicircular canal function, as measured by the threshold

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A systematic comparison between the resp... persons with bilateral loss of labyrinthine fun... force environments has demonstrated that the... essential to the causation of typical sympt ms ... biel *et al* 1960 Meek *et al* 1962 Johns n *et al* 1963) Even a partial loss of the vestibular func... tion and among those so protected are patients ... mycin sulfate for the treatment of Ménière's d... administered streptomycin sulfate to eight pat... in amounts just sufficient to result in unstead... vestigation no one suffered a loss in hearing ... for the vestibular system Ten years later ... examined none experienced symptoms of m... Rotation Room and their hearing when c mp... the treatment had improved in three and was ... *et al* 1965 *a*) These findings suggested the ... mycin sulfate might suppress the vestibular f... ficiently to prevent motion sickness and ... margin of safety Moreover in none of the re... this drug in the treatment of Ménière's disease ... *et al* 1949 Ruedi 1951 Hanson 1951) was ... impairment of hearing

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## METHOD

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caloric test (McLeod & Meek, 1962) and susceptibility to vomiting in the Slow Rotation Room (SRR) (Meek *et al*, 1962, Johnson *et al*, 1962).

The threshold caloric test (TCT) was performed in a darkened room. The animal's head was secured and positioned so that the horizontal semicircular canals were in a vertical plane. The ear was irrigated with about 70 cc of water in a period of 40 seconds. Beginning with a temperature of 35.0°C, or lower, depending on previous results, the temperature of the water was dropped in successive tests until nystagmus was observed by electronystagmography, Frenzel goggles, and dim illumination. Hearing tests were not performed.

In determining the susceptibility to emesis, the animal was exposed to rotation in the SRR while free to move about in a small cage and under good illumination. Initially, an attempt was made to obtain a "threshold angular velocity" which led to emesis within a period of 10 minutes. This often required so many trials that there was a risk of adaptation, and a single angular velocity of 10 rpm was used thereafter. Inasmuch as the strength of the physical stimulus to the canals involved head rotations which were not experimenter paced as well as angular velocity of the SRR, not much significance was attached to differences in the "duration" of exposure. Premonitory symptoms such as crouching, chewing, and especially retching were probable indications of canal sickness, however, the sole criterion of positive canal sickness was emesis.

An attempt was made to estimate the degree of ataxia in some instances (six out of eight). This was done by conditioning animals to walk a rod or wire, the procedures, however, were in the developmental stage and few quantitative data were obtained.

The streptomycin sulfate was given in courses. Depending mainly on body weight, the initial course consisted of 16 to 23 doses of 50 mg each given intramuscularly once daily. If at about the end of one month following the last dose there was no sign of canal suppression, additional courses were given until the desired result was obtained, factors in addition to body weight sometimes influenced the number of doses to be given. The clinical tests were performed repeatedly, both during the administration of the drug and for a period of at least six months thereafter.

Subsequently, the animals were sacrificed by means of intravital cardiac perfusion with Heidenhain-Susa fixative. The temporal bone block which included both inner ears was dissected out from the skull and immersed in the same fixative for a further period. After completing the fixation the bone was decalcified in 5% trichloroacetic acid solution. The end point of decalcification was chemically detected with 5% ammonium oxalate and 5% ammonium hydroxide mixture. Dehydration was done in 30%, 50%, 70%, 80% (with iodine solution in it), 95%, 100% of ethyl alcohol, and ether-100% ethyl alcohol mixture (1:1). The specimen was processed to 3% celloidin (2 weeks), 6% celloidin (2 weeks), and 12% celloidin (3 weeks). The celloidin hardening process was done very slowly. The bone

was sectioned serially in a horizontal plane at 20 microns One of each ten sections was stained in Hematoxylin-Phloxine, mounted on glass slides and examined by light microscopy

The pathological findings of both vestibule and cochlea were portrayed using a graphic reconstruction technique (Guild 1921, Schuknecht, 1953).

## RESULTS

### *Case Reports*

*Case EP*

This animal received one course, total 1100 mg of the drug Before medication, emesis occurred at SRR velocities of 3 and 4 rpm, TCT values were 34 5°C R (right) and 35 0°C L (left), and the animal could walk a 1/4′ rod without difficulty Thirteen days after treatment began the TCT value was 34 0°C both ears, with a gradual fall to 31 5°C R and 31 0°C L on the last (22nd) day at which time the animal was grossly ataxic Four days after treatment there was no response to irrigation with temperature of water at 15°C, and there was no response at 10°C three months later Approximately four months after medication the TCT values were 20 0°C R and 10 0°C L, indicating a slight return of function, but there were no manifestations of canal sickness with SRR velocity at 10 rpm, the animal was somewhat ataxic on walking and could not cross the 1/4′ rod No further clinical tests were carried out until prior to sacrifice 10 months following the medication, when the TCT value was 35 0°C both ears There was no clear evidence of ataxia, although the animal was "uncooperative' on the

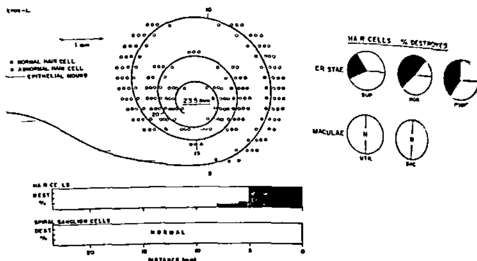



FIG 1 Graphic portrayal of hair cell loss in inner ear end organs ... streptomycin sulfate In the circles portraying the cristae the horiz... the division between upper and lower half (Case EP)

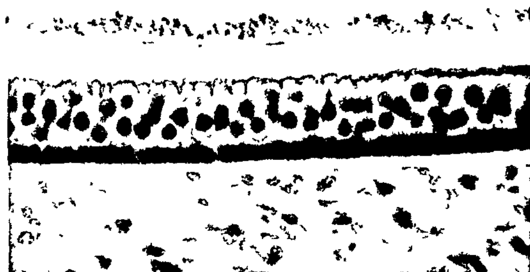


FIG 2 Microphotograph showing the intact macula sacculi from the ear of the animal which has received 1100 mg of streptomycin sulfate (Case FP) ×840

27/1000" wire, vomiting did not occur after 10 minutes of rotation in the SRR at 10 rpm

Pathological studies revealed severe damage to the cristae, some hair cell loss in the basal turn of the cochlea, but no evidence of injury to the maculae (Figs 1 and 2) The sensory epithelium of the summits of the cristae showed the severest pathological changes and hair cell loss of any in the present series (Fig 3)

The striking features of this case were 1) the slow but eventual functional recovery, except loss of susceptibility to canal sickness, in the presence of severe loss of hair cells in the cristae, and 2) the absence of hair cell loss in the maculae

*Case FC*

This animal received one course of the drug, 1150 mg Before medication the TCT values were 33 3°C R and 33 0°C L, and the animal could easily cross the 1/4" rod Vomiting did not occur at a SRR velocity of 5 rpm The exposure at 10 rpm was not done After 950 mg of streptomycin, the TCT values were 33 0°C R and 33 4°C L, but one day after the last injection the value was 28 4°C in both ears Thereafter, a rapid change occurred, and eight days following the last injection there was no response at an irrigating temperature of 7°C Parallel to the fall in TCT values there was increasing ataxia, and at time of complete suppression, the animal could

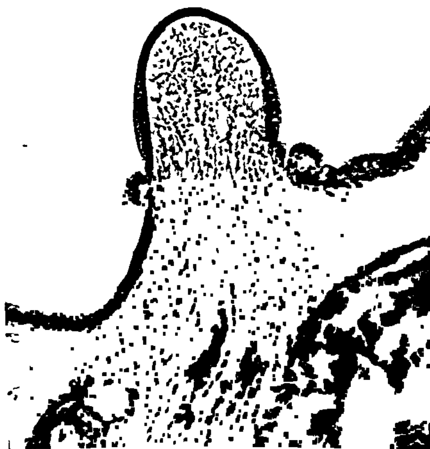


Fig 3 Microphotograph showing a view of the severe end organ pathology in the crista of a horizontal semicircular canal Note that the hair cell damage is more prominent on the summit of crista (Case F1 ) ×1 5

not walk the 1/4 rod and crossed the 1 2" rod with difficulty Clinical tests were not carried out between the 8th and 74th day after medication at which time the TCT value was 27°C L but there was no response (11°C) on the R Three weeks later there was a response on the right at 10°C and the animal could walk the 1/4" rod Six months after medication the TCT values were 25 8°C R and 26 2°C L Ten months after medication the TCT value was 35 0°C both ears and the animal could walk the 27 1000" wire Emesis did not occur at 10 rpm in the SRR

Pathological findings revealed moderate loss of hair cells in the cristae and cochlea but none in the macula

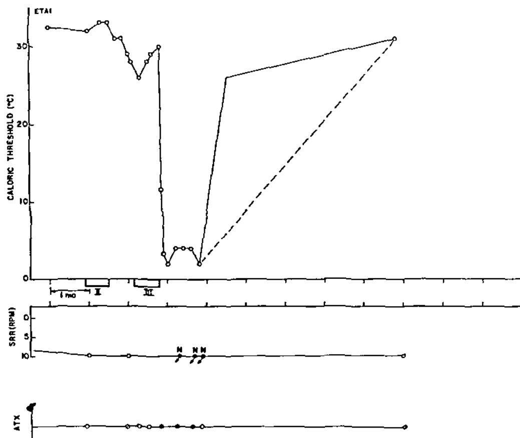



Fig 4 The course of caloric threshold and susceptibility to canal sickness in another monkey after 3000 mg of streptomycin sulfate (Case ET) White circles in the caloric test column indicate the testing points White circles in the Slow Rotation Room column indicate the positive vomiting and black dots with N and an arrow indicate no emesis at that level (10 rpm) White circles in ataxia test column indicate the normal rod walking ability White circles with diagonal lines indicate slight ataxia and black dots indicate severe ataxia The arabic numbers and rectangles below horizontal lines with one month step abscissa, indicate the numbers of streptomycin course

Noteworthy features of this case were 1) the long (more than six months) recovery period following canal suppression, and 2) the absence of any morphological damage in the maculae

*Case ET*

This animal received three courses of the drug 950 mg, 1050 mg and 1000 mg, respectively Prior to medication the TCT values were 33 7°C R and 33 8°C L Emesis occurred at SRR velocity of 3 rpm, and the animal could readily cross the 1/4 rod After one course of medication there was only slight canal suppression (31 0°C R and 31 0°C L), emesis occurred at a SRR velocity of 7 5 rpm, and the animal fell once (abnormal) while walking the 1/4 rod

The second course (1050 mg) was begun three months after completion [illegible] at which time the animal had apparently recovered, although

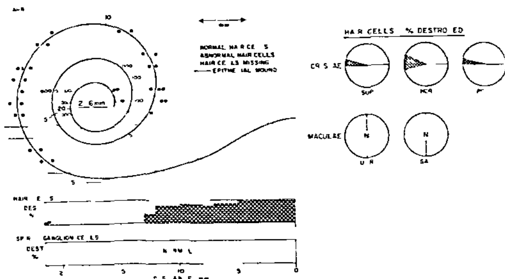



FIG 5 Graphic reconstruction demonstrating slight vestibular end organ pathology and moderate cochlear hair cell loss after 3000 mg of streptomycin sulfate (Case ET)

the caloric test value revealed a slightly raised threshold Following medication the TCT value gradually decreased, and 20 days afterward was 25 2°C R and 27 0°C L The animal failed to walk the 1/4 rod but could walk the 1″ rod Emesis however, still occurred at SRR velocities of 10 rpm

The third course (1000 mg) was begun three weeks after the second and on the 16th day of medication the TCT temperatures had fallen to 30°C R and 29 2°C L but the animal could walk the 1/4 rod On the first day after the completion of the course there was no nystagmus response with an irrigating temperature of 11°C and the monkey was grossly ataxic Five days after the course there was no nystagmus response at 1°–2°C and this value was unchanged (1°–3°C both ears) even one month after the last injection During this period the monkey did not vomit in the SRR at 10 rpm and failed to cross the 2 1/2 rod Six months after the last course the TCT values were 30 0°C R and 31 0°C L The animal could cross all but the 1/4″ rod without difficulty and emesis occurred at 10 rpm in the SRR The clinical findings are summarized in Fig 4

Pathological findings are summarized in Fig 5 and it is seen that there was little hair cell pathology in the cristae but moderate loss in the cochlea The maculae were intact morphologically

The interesting features in this case were 1) the relatively slight loss of hair cells in the cristae after three courses of streptomycin sulfate (3000 mg), yet complete suppression of canal function after completion of the third course as indicated by the TCT 2) long suppressed period (for about one month) and 3) the relatively greater damage to the cochlea than to the cristae

*Case ES*

This animal received three courses of the drug 850 mg, 1000 mg, and 1150 mg Prior to medication the TCT temperature was 34 5°C both ears, emesis occurred at 5 rpm in the SRR, and there was no ataxia During medication with 850 mg and for a month thereafter the maximum canal suppression as indicated by the TCT temperature was 33 5°C both ears Ataxia did not appear and the animal vomited at 10 rpm in the SRR

The second course (1000 mg) was begun three months after completion of the first one There was only a slight further suppression during and for three weeks after medication as indicated by the TCT (31 0°C both ears) Third course was begun without further delay in the hope that there might be cumulative effects

During administration of the drug in the third course (1150 mg) there was no evidence of canal suppression, and two days before completion the TCT temperature was 31 0°C both ears Twelve days after medication the TCT temperatures had fallen to almost their lowest values (22 3°C R and 23 0°C L), the monkey did not vomit at 10 rpm in the SRR, but there was no definite evidence of ataxia This was the only occasion the animal



Fig 6 Microphotograph showing very slight hair cell pathology in the horizontal semicircular canal of S a after 3000 mg of streptomycin sulfate injection (Case ES) × 150

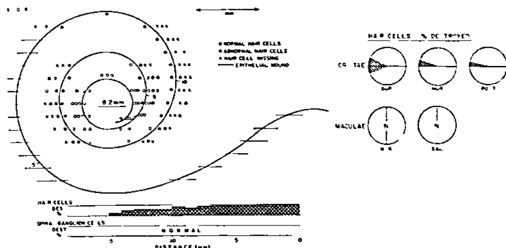



FIG 7 Graphic display showing slight canal end organ pathology after 3000 mg of streptomycin sulfate (Case ES) The cochlear pathology was severe

*did not vomit out of a total of eight previous and five subsequent exposures* The TCT temperatures slowly rose, about one month later they were 25 0°C R and 26 0°C L, five months after medication the temperature was 35 0°C both ears, the animal vomited in 55 seconds on exposure in the SRR and could walk the 27/1000" wire

Pathological findings revealed very slight hair cell pathology in the cristae (Figs. 6 and 7) but severe loss in the cochlea (Figs 8 *A* and *B*) There was no evidence of damage in the maculae or spiral ganglion

Three things were demonstrated in this case 1) the partial suppression of canal function sufficed to abolish canal sickness, 2) the cochlea may be far more susceptible to the ototoxic effects of streptomycin sulfate than the cristae, and 3) a shorter interval between courses may increase toxic effect

*Case FN*

*This monkey received three courses of streptomycin sulfate 1050 mg* 1150 mg, and 1150 mg, respectively During and after the first course, there was no definite change of canal sensitivity as indicated by TCT value, the animal vomited in the SRR, and there was no evidence of ataxia

A second course (1150 mg) was begun three months after completion of the first, again with no definite evidence of canal suppression, so, a third course was begun 19 days after completion of the second The TCT temperatures before this course (1150 mg) were about 34 0°C R and 33 0°C L (strong) Two days before completion of the course the animal could walk the 1/4" rod

The first day after completion of the third course (1150 mg) gross ataxia appeared but the TCT temperatures were still 30 0°C R and 31 6°C L.




FIG 8 A M cropl otograph showing normal organ of Corti from upper middle and upper basal turns in a normal squirrel monkey × 185

The fifth day afterward the TCT temperature was 2 0°C L and no response on the right with 2°C water irrigation On the eleventh day TCT value was 2 8°C both ears (Fig 9) At this time emesis did not occur in the SRR at 10 rpm and the monkey could barely cross the 2 1/2 rod with a shuffling and unsteady gait The complete canal suppression was short lived The TCT temperatures were 20 0°C R and 22 0°C L on the 18th day after the course but vomiting did not occur in the SRR at 10 rpm and




FIG. 8 B Microphotograph demonstrating the partial outer hair cell loss in upper middle turn and the total loss of outer hair cells with some supporting cell destruction in the upper basal turn after 3000 mg of streptomycin sulfate injection (Case ES) × 120

the animal remained slightly ataxic The rod-walking ability improved and the animal could cross the 1/4" rod with some difficulty on the 25th day. The TCT temperatures gradually rose and were 26 0°C R and 27 0°C L on the 36th day, the animal vomited, for the first time after medication, on the 37th day Six months after the last course the TCT value was 35 0°C both ears, emesis occurred on rotation at 10 rpm, and the animal could walk the 27/1000" wire.

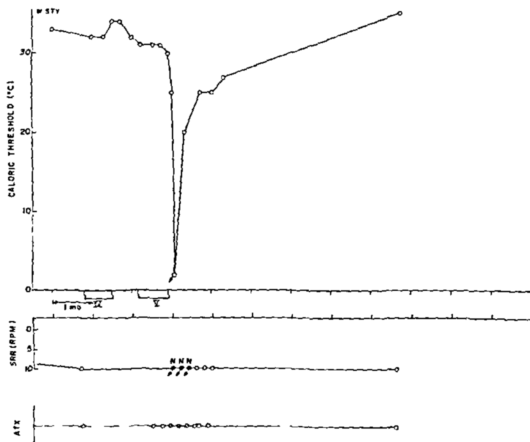



FIG 9 The course of caloric threshold susceptibility to emesis in Slow Rotation Room, and ataxia after 3350 mg of streptomycin sulfate (Case FV) The hair cell pathology in cristae was moderate, very slight in maculae, and moderate in cochlea with no spiral ganglion cell lesion

Pathological study revealed moderate end organ pathology both in cristae and cochlea The maculae were almost intact morphologically (Fig 10)

The most striking features of this case were 1) the initial appearance of gross ataxia at a time when the TCT value had decreased only slightly, and the disappearance of ataxia before there was a susceptibility to canal sickness, and 2) the possible cumulative toxic effect of the drug when a relatively short interval (19 days) separated two courses

*Case DH*

This animal received four courses of the drug 900 mg, 700 mg, 300 mg, and 900 mg Prior to the medication the TCT value was 34 0°C both ears, and the animal vomited at 3 rpm in the SRR, ataxia tests were not carried out During and after the first course of medication there was no change in susceptibility to canal sickness, and about three months later a second course was begun

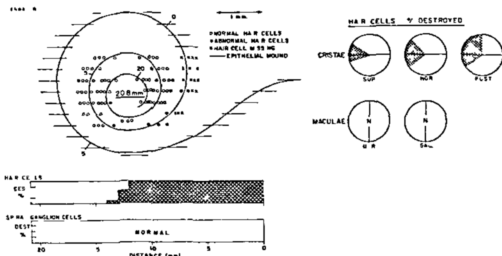



Fig 10 Graphic reconstruction showing moderate pathology both in cristae and in organ of Corti after 3350 mg of streptomycin injection (Case FY)

The second course (700 mg) succeeded in suppressing canal function only slightly (28 5°C R and 27 5°C L), and inasmuch as there was no change in susceptibility to canal sickness, a third course was begun about two months after completion of the second

The third course (300 mg) succeeded in lowering (maximally) the TCT temperatures to 27 0°C R and 26 4°C L on the eighth day after treatment, but the animal did not lose its susceptibility to canal sickness at 10 rpm and could walk the 1/4" rod without difficulty The last TCT was carried out two months after the last injection and was 29 8°C both ears

The fourth course (900 mg) began about four months after completion of the third, and after two weeks the TCT temperatures were 27 8°C R and 18 0°C L, and the animal was ataxic but vomited at 10 rpm in the SRR On the last day of medication the TCT temperature was 24 4°C both ears and thereafter gradually fell to its lowest level, 20 0°C both ears, on the 24th day post medication At this time the animal was ataxic, crossed the 1/4" rod with difficulty, and did not vomit at 10 rpm in the SRR Thirty days post-medication there was a significant increase in the TCT temperature on the right (26 0°C) but only a slight increase on the left (21 4°C), the animal was still ataxic and did not vomit in the SRR Thereafter, there was a gradual loss of the ataxia, and six months post-medication the animal could cross the 27/1000" wire, the TCT temperature was 32 0°C both ears, but susceptibility to canal sickness had not returned

The hair cell loss was moderate in the cristae, slight in the maculae (Fig 11), however, both the organ of Corti and spiral ganglion were almost completely destroyed (Fig 12)

The noteworthy features in this case were 1) the large amount of the



Fig 11 Microphotograph showing the saccular macula with a slight change after 2800 mg of streptomycin sulfate (Case DH) Hair cell population is normal ×840

drug which was enough to greatly damage the cochlea that was needed to suppress canal function, and 2) the comparative effects on ataxia and susceptibility to canal sickness

*Case DR*

This animal received four courses of streptomycin sulfate, 900 mg, 1150 mg, 900 mg, and 1100 mg. Prior to administration of the drug the TCT temperature was 34.0°C both ears, and emesis was observed at 5 rpm in the SRR. Both during and after the first course the animal vomited in the SRR, and three months afterward the second course was begun

In the second course (1150 mg) the TCT temperature fell to 29.9°C both ears nine days after the last injection, but ataxia was not observed, and the animal vomited in the SRR.

The third course (900 mg) was begun 50 days after completion of the second Shortly before this time the TCT temperatures were 32.2°C R and 32.8°C L; the animal was not ataxic and vomited in the SRR. During and after this course, the animal was not ataxic and continued to vomit in the SRR; the lowest TCT temperatures were 28.2°C R and 27.5°C L recorded on the 18th day post-medication

[illegible] course (1100 mg) was begun 3 1/2 months after the third, at which tim[illegible] TCT temperature was 29.0°C both ears The maximum

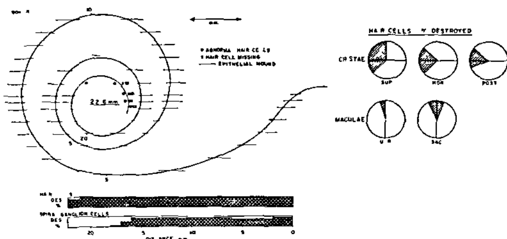



Fig 12 Spiral display and bargrams demonstrating the severest destruction of cochlear hair cells and spiral ganglion cells after 2800 mg of streptomycin sulfate injection in the present series of monkeys (Case DH) The hair cell pathology in cristae was also moderate The caloric threshold was suppressed as low as 20 C, and recovered This animal did not show any recovery in canal sickness susceptibility with emesis when tested however

canal suppression occurred between the 5th and 12th days after the last injection and the TCT temperature (both ears) was about 24.0°C, during this period the animal did not vomit at 10 rpm in the SRR Fifteen days after the course the TCT temperatures were 30.0°C R and 28.0°C L, the animal vomited in the SRR and could walk the 1/4 rod without difficulty The 36th day after completion of the fourth course the TCT temperatures

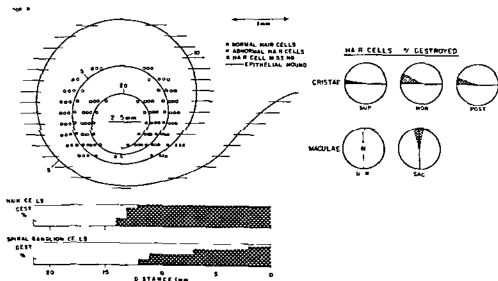



Fig 13 Graphic reconstruction showing very slight vestibular end organ pathology after 4050 mg of streptomycin sulfate (Case DR) The highest caloric threshold obtained was 24°C Both organ of Corti and spiral ganglion had moderate pathology



Fig 14 Microphotograph demonstrating the entire cochlea with the moderate pathology in organ of Corti and spiral ganglion after 4050 mg of streptomycin sulfate injection (Case DR) Notice the loss of primary neurons in osseous spiral lamina in the basal turns Organ of Corti and spiral ganglion are morphologically intact in middle and apical turns × 30

were 31 8°C R and 32 0°C L The animal was not tested again till prior to sacrifice seven months later at which time the TCT temperature was 35 0°C both ears The animal could cross the 27/1000″ wire and became sick at 10 rpm in the SRR

*Pathological studies revealed slight pathology both in cristae and* maculae, and moderate pathology in cochlea (Fig 13) The low magnification view of the entire cochlea clearly demonstrates the different degrees of pathology in each turn (Fig 14)

This case seemed to establish the fact that a long period of time was required to restore the normal TCT values The canal sickness was not abolished until the TCT temperatures, shortly after medication, fell below 27 0°–28 0°C

*Case DM*

This animal received five courses of streptomycin sulfate: 800 mg, 800 mg, 800 mg, 1000 mg, and 1200 mg (total 4600 mg) Prior to medication the TCT temperature was 34 0°C both ears, and emesis occurred at 3 rpm in the SRR The first four courses neither abolished susceptibility

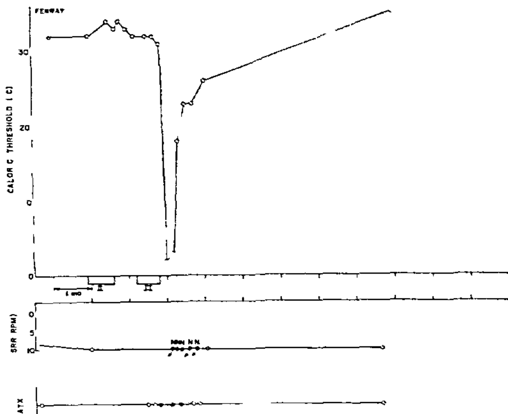



FIG 15 The course of caloric threshold change ataxia and canal sickness susceptibility change The monkey had very slight crista lesions with moderate damage to organ of Corti after 4600 mg of streptomycin sulfate (Case DM)

to canal sickness nor caused ataxia the lowest TCT temperature was recorded shortly after completion of the fourth course and was about 31 0°C both ears

The fifth course (1200 mg) was begun three weeks after completion of the fourth at which time the TCT temperature was about 31 0°C both ears On the second day after the last injection the TCT temperatures were 24 0°C R and 27 0°C L and the animal was slightly ataxic and failed to vomit in the SRR Complete suppression occurred between the second and eighth day when there was no response at a temperature of 3°C (Fig 15) Fifteen days after medication the TCT temperatures were 20 0°C R and 23 4°C L and the animal was still ataxic and did not vomit in the SRR Twenty days afterward the TCT temperatures were 23 0°C R and 21 8°C L and when tested the following day the animal could walk the 1/4 rod without difficulty and became sick at 10 rpm in the SRR Six months after completion of the fifth course the TCT value was 35 0°C both ears emesis occurred at 10 rpm in the SRR and the animal could walk the 27/1000 wire

mechanostructural differences between summit and slopes. One difference has been emphasized by Nomura *et al.* (1965) who demonstrated that the efferent vestibular fibers ended in the slopes and not in the summits of the cristae in guinea pigs. It is explained in part at least by the findings of Wersäll & Hawkins (1962) who found that type 1 hair cells which were more commonly located on the summit (Wersäll, 1956), were more easily damaged than type 2 by chronic streptomycin intoxication in cats.

The degree of damage to the cristae varied from very slight to severe and had no relation to the number of courses of the drug given, indicating great individual variance in susceptibility to the drug. There was only slight variance in predilection of the toxic effect among the three canals; this has special significance when correlating the findings with the threshold caloric test which involved primarily the horizontal canals.

The clinical findings at time of sacrifice were essentially unchanged when compared with values obtained prior to the administration of the drug in five of the eight monkeys in which the pathological changes in the cristae varied from very slight to moderate. Among the remaining three, EP no longer manifested canal sickness although the caloric test revealed a normal threshold value; ET had a slightly raised (3.1°C) caloric threshold, manifested slight ataxia, but susceptibility to canal sickness had returned; DH had a slightly raised caloric threshold (2.0°C), was not susceptible to canal sickness, but did not manifest ataxia.

The possible implications of these findings for man are important. If they hold true, then the threshold caloric test is not necessarily reliable as an indicator of normal morphology of the cristae, although quite reliable as an indicator of normal function in terms of susceptibility to canal sickness and ataxia.

With regard to the maculae none of the abnormal clinical findings at time of sacrifice could reasonably be ascribed to these organs. The pathological alterations noted in the maculae were very slight in four instances, three in the saccular maculae and only one in the utricular macula. Among the latter only DH manifested clinical abnormalities; the raised threshold to caloric stimulation was surely referable to the canals, and the loss of susceptibility to emesis in the SRR almost certainly was of the same origin.

This relative freedom from injury in the maculae was striking and suggests that, with judicious use, streptomycin sulfate combined with subsequent pathological confirmation has an important place in carrying out functional vestibular experiments in squirrel monkeys.

Pathological changes were found in the organ of Corti in all eight monkeys and in four of these there were also alterations in the spiral ganglion. There were very minimal pathological changes in the stria vascularis in a few instances. These findings are similar to those reported by McG[illegible] Olszewski (1962) on cats which received streptomycin sulfate [illegible]0 200 [illegible] daily, total 1800–5600 mg); pathological studies revealed

cochlear alterations in three of seven animals and in two of these three spiral ganglion cell lesions also were present

Our findings insofar as they are extrapolable to man interdict the use of streptomycin sulfate for the prevention of motion sickness In the case of EP where the predilection for injury to the cristae was greatest and for the organ of Corti least the possibility existed that a smaller dose of the drug might have had the desired effect without injuring the cochlea At the other extreme, DM required five courses of medication which resulted in only very slight damage to the cristae but moderately severe damage to the organ of Corti

*Findings prior to sacrifice*

In view of the findings just described the question must be raised whether pathological changes existed at the time the animals were originally selected for experimentation This possibility cannot be discounted although 11 other animals with normal caloric thresholds and high susceptibility to canal sickness have been sacrificed and none revealed 'spontaneous pathological changes in the vestibular organs (Graybiel *et al* 1965 *b*) Moreover in the present series not only was an ototoxic drug administered to the point of canal suppression but also three of the eight had functional abnormalities at time of sacrifice

The most striking features in this period under discussion are 1) the individual variance in susceptibility to the toxic effects of streptomycin sulfate and 2) the time course of the clinical changes There was a small margin between the fatal dose and the amount of the drug necessary to suppress canal function a number of monkeys receiving slightly larger daily doses than those used in this series did not survive This led to the administration of the drug in courses Here it was learned that in order to ensure a cumulative effect the second course had to be instituted within a short time preferably not delayed longer than two weeks following completion of the preceding course It would appear that the squirrel monkey is far less susceptible to the vestibulo toxic effects of streptomycin sulfate than man

With regard to the clinical indications of the ototoxic effects there was much similarity in terms of the appearance and subsequent disappearance of these abnormal signs This was not surprising inasmuch as these effects had their genesis largely if not entirely in the semicircular canals The clinical effects were never prominent until nearly all of the drug had been administered and the maximum effects were usually noted within a few days after the last injection A slight increase in the caloric threshold was usually noted first then a further increase accompanied by ataxia and loss of susceptibility to canal sickness

At the time of maximum suppression nystagmus was not obtained on irrigation with cold water in five instances and the threshold temperatures were 20° 24°C in the other three At the latter levels susceptibility to

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OTO-LARYNGOLOGICA

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# 40 YEARS COLLEGIUM OTO-RHINO-LARYNGOLOGICUM *AMICITIAE SACRUM*

BY

EELCO HUIZINGA

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM O

PRINTED IN SWEDEN BY

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UPPSALA 1966

40 YEARS COLLEGIUM
OTO RHINO-LARYNGOLOGICUM
AMICITIAE SACRUM
(1926 1966)

Written at the place of foundation of the Collegium O R L A S by EELCO HUIZINGA who attended all the meetings of the Collegium as one of the best things in his life

# INDEX

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# FOREWORD

For a scientific Society 40 years is an important age It means that a complete generation is replaced by its successors Famous names have become memories and young people are showing the results of the work that may bring them renown and glory Our fathers in science have shown us the fruit of their experience and we go on cultivating the land

Eelco Huizinga is, I think, the man of choice to tell us about this period of 40 years during which he was a founder, a member, the general secretary and now a honorary member of the Collegium O R L A S

In his „40 years Collegium Oto Rhino Laryngologicum Amicitiae Sacrum" we shall meet many old friends and new ones, old topics and new ones, but above all one continuous line of good companionship and an excellent field for the growth and exchange of knowledge

L B W Jongkees
General Secretary of the Collegium O R L A S

## INTRODUCTION

At the business meeting of the Collegium O R L A S at Würzburg in 1964 it was decided to write its History. This is an excellent idea and it is the right time to do it since at this moment some of the founders are still alive. But it is not an easy, and certainly it is a very extensive task, as the history of the Collegium is closely connected with the history of oto-rhino-laryngology, which has undergone so many important changes during the last forty years. But there is so much more to record. Why was the Collegium founded in the Netherlands? Who have contributed most to its foundation? How where the meetings arranged and are they still the same? Have the subjects, discussed at the meetings, undergone any important changes? What was the influence of the war upon the Collegium? It seems worth while to describe all this. The younger generation may be interested in it. It is surely of use to them to hear about the great names in their profession and to take cognizance of the evolution of oto-rhino-laryngology. It is one of the most important sections of medicine, but it is a universal complaint that its history is often neglected.

In his famous novel ' South Wind ' NORMAN DOUGLAS writes the following: "History deals with situations and figures not imaginary but real. It demands therefore a combination of qualities unnecessary to the poet or writer of romance — glacial judgment coupled with fervent sympathy. The poet may be an inspired illiterate, the romance writer an uninspired hack. Under no circumstances can either of them be accused of wronging or deceiving the public, however incongruous their efforts. They write well or badly, and there the matter ends. The historian who fails in his duty deceives the reader and wrongs the dead. A man weighted with such responsibilities is deserving of an audience more than usually select."

Well, from the beginning, the Collegium has been a "more than usually select audience". This heightens the effort not ' to deceive the reader and wrong the dead '. Anyhow there is at least "the fervent sympathy", there is even more. It is *Collegium* Oto Rhino-Laryngologicum *Amicitiae Sacrum*. The last part has always been of the utmost importance. Friendship and above all international friendship is the only way to a better world. The Frenchman LACORDAIRE once stated "L'amitié est le plus parfait des sentiments de l'homme" ('Friendship is the most perfect of human feelings"). And to continue in the "French vein ' one can add that its value — like that of good wine — increases with age. Therefore it seems a good thing that one of the older members should write this history, one who has met with much friendship in and through the Collegium.

## THE INITIATION

During the summer of 1926 a letter was sent to a limited number of ear specialists in 14 countries of Western Europe on the initiative of BENJAMINS, in his name and in that of his friend DE KLEYN. It read as follows: "At the meetings of the Oto-Rhino-Laryngological Societies and at the International Congresses the subjects dealt with are of widely divergent character. The purely scientific subjects often do not receive sufficient attention chiefly on account of the following reasons:

1. On account of the great number of papers read one's attention is distracted and too short a time is allowed for each speaker.

2. On account of the presence of a number of members whose interest in purely scientific subjects is only slight.

We have therefore decided to invite a number of colleagues whom we know to take an interest in scientific problems to start an international "Collegium Oto-Rhino-Laryngologicum" in which purely scientific problems will be discussed. These may be of a theoretical, experimental, or clinical character, provided that the purely scientific aspect of the problem is predominant. The membership of such a society must necessarily be limited, the number of papers read must also be limited in order to allow at least half an hour for each paper. In some instance as much as an hour might be advisable in which case consent from the board would have to be obtained.

In this way it will be possible to pay due attention to each problem and the presence of a number of people working in the same direction should promote a very fertile discussion.

The language used would be English, French or German so that the majority of those present would be able to understand each other, thus we think that it would be possible to invite representatives from the following countries: Austria, Belgium, Czecho-Slovakia, Denmark, Finland, France, Germany, Great Britain, Holland, Hungary, Norway, Spain, Sweden and Switzerland.

We should be greatly obliged if you would send us the names of others in your country who are in your opinion suitable to receive an invitation. We should like to remind you here that limitation in the number of members is advisable. If we find sufficient response among our colleagues we intend to convoke a meeting in Groningen in October or November of this year at which a scheme worked out by us on the above lines and previously circulated will be discussed, the Society properly constituted and a number of scientific lectures delivered.

If the project proves successful, meetings might be held yearly, each time in one of the participating countries."

This letter produced an immediate response and as a consequence it was possible to have the first meeting of the Collegium at Groningen in 1926 (from October 8-10th) with ZWAARDEMAKER in the chair.

The initiative of BENJAMINS and DE KLEYN was a very successful and also a very special one Living at the present time with international symposiums and round table conferences every day, one can hardly imagine that it was a very original idea in 1926 to organize a meeting of a number of carefully selected specialists for a purely scientific discussion of ear, nose and throat problems The excellent reception given to this idea was due largely to the world wide prestige of BENJAMINS and DE KLEYN, both as men of science and as personalities

C. E BENJAMINS (1873 1940) (fig 1 ) was a man endowed with many talents Thanks to his very active mind he has worked in nearly all the various fields of

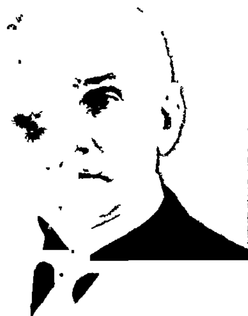


Fig 1 C. E Benjamins

oto rhino laryngology He was one of the first in Europe who saw the importance of allergy for our specialty He was a master in microscopy (crista quarta, tumeurs osseuses du nez et des sinus, a new form of nasal tumour) but also in physiolog ical investigation

As a matter of fact he became assistant of the famous physiologist ZWAARDEMAKER in Utrecht, where he put up his plate as an ear, nose and throat specialist in

1911 Before that time he had been an oto rhino laryngologist in Semarang, after his studies under JURASZ, KILLIAN and LUC The chapter on tropical diseases written by BENJAMINS for DENKER and KAHLER's textbook was at that time judged to be one of the bests in this domain In 1924 he was appointed a professor at Groningen University As the first general secretary of the Collegium he was its motive force till 1940

A P H A DE KLEYN (1883 1949) (fig 2) received his training from QUIX, (professor of otology in Utrecht) and later in Vienna, which at the time was the Mecca of Oto Rhino Laryngology For many years a specialist in the USA was



Fig 2. A P H A de Kleyn

hardly accepted unless he bore the Vienna stamp — Today, everybody goes to the USA — DE KLEYN maintained close relations with many of the famous men from the POLITZER school, several were later member of the Collegium He had an exceptional knowledge of the physiology of the vestibule, on which subject he delivered several communications Apart from numerous experiments in

mammals he published many papers on human physiology and pathology (various forms of nystagmus, etc) In his days he was accepted as the foremost authority on neuro otology In 1936, after a few refusals, he accepted a professorship at Amsterdam University, as successor to BURGER In 1926 existed still the wellknown cooperation with MAGNUS, professor of pharmacology in Utrecht, who died in 1927

H ZWAARDEMAKER (1857 1930) (fig 3) became professor of physiology at Utrecht University in 1897 Prior to this, he had as a military medical officer trained in otology under SCHWARTZE and POLITZER In his early years he had been a physiology assistant, and he had always maintained relations with physiology



Fig 3 H Zwaardemaker

In this he was encouraged by the great DONDERS, who was deeply interested in his pioneer work on olfaction ZWAARDEMAKER exerted a considerable influence on the scientific standing of O R L in the Netherlands, for many years, all university professors in this field in the Netherlands have been his pupils, directly or indirectly Figure 3 is a reproduction of the painting made when he took his leave It depicts the four main subjects of his interest viz radioactivity, olfaction, acoustics and phonetics No less than three of these concern our field, and without doubt they represent his best work With regard to the ear, he made striking predictions concerning spectacles for the deaf" there was also his study, jointly with QUIX, of whispered speech with an evaluation of high and low pitched sounds which we tend to forget in these days of ever more complex apparatus The first quantitative investigations into the vestibular rotation reaction were made under his direction

## 'Locarno Scientifique"

The attendance of the first meeting was highly satisfactory Of 58 physicians who had expressed themselves in favour of the suggestion made by Benjamins and De Kleyn, 34 were present in Groningen They came from 10 different countries (9 from the Netherlands, 5 from Denmark and Germany, 4 from England, 3 from Sweden, 2 from France, Austria and Switzerland and 1 from Czecho-Slovakia and Hungary) Italy was conspicuously absent on this first occasion, the contact, which later was to become so intensive, had apparently not yet been made Members from Finland, Norway and Spain did not attend, and Belgium had declined the invitation for the time being This was eight years after World War I

The attitude of the medical profession to war is a very special one The two world wars with their massed armies were made possible by the evolution of medicine But physicians must continue their duties and, in view of the Red Cross idea which does not discriminate between friend and foe, they must be above the course of events But war ruins so much in human interrelations that medical feelings, too, are subject to change In 1926 it was still difficult to arrange a meeting of people who had been enemies in the great war, at least a meeting on scientific, friendly terms The meeting in Groningen was probably among the first — if not the very first — of its kind, certainly it was the first meeting of otologists

Objections, as expected, arose chiefly from Western countries which had suffered most from the war Belgium and France Fortunately, two Frenchmen were nevertheless present There was brilliant Georges Portmann from Bordeaux, who from the start played an important role in the Collegium, there was Mouret from Montpellier – the investigator of the anatomy of the petrosal bone

From Germany came the wellknown clinician Voss, his pupil Grahl, Albrecht, Wittmaack (whose original ideas and numerous animal experiments greatly inspired O R L research) and his pupil Eckert Mobius – an authority on microscopy Of the Germans, Voss from Frankfurt was the leading figure He was an exceedingly kind and good man, and kindness and goodness are important character traits in a physician The most remarkable man of the Germans was without any doubt the very scientific Wittmaack He was often aggressive, his best pupil Eckert Mobius called him once "a pike in a carp-pond" But he was a good member of the Collegium in the first years During the banquet of the German O R L meeting in Hamburg he mounted suddenly on a chair and deeply moved he made the following speech "I am accustomed to the fact that I am not understood but now I insist to be heard and to be understood well For the first time after the war we have two distinguished Dutch guests, De Kleyn and Benjamins We are very grateful that they are present here and we hope that this will be the beginning of a new era of scientific collaboration with the colleagues of the whole world " These words which made a great impression, were spoken in May 1926, the Collegium started in October

None of those present will ever forget Portmann's dinner speech, it was

perhaps the best he ever made, and that means something! Recalling a pleasant conversation with Voss, he referred to an interview which shortly before had taken place between BRIAND and STRESEMANN This was the time of the rapprochement between France and Germany (l'histoire se repète), and the discussions between the two statesmen at Locarno had been productive Said PORTMANN in Groningen we have had "une Locarno scientifique"

ARISTIDE BRIAND was among the greatest politicians France ever produced The Paris memorial in honour of this statesman bears an inscription recalling one of his statements about war, which also has a bearing on medicine It reads „Les savants luttent chaque jour pour nous preserver de la tuberculose, du cancer et la guerre, l'horrible guerre serait le seul mal contre lequel l humanite se declarerait impuissante? Je ne veux pas le croire "
("Every day, scientists fight to protect us from tuberculosis, from cancer, and should war — horrible war — be the only disease against which mankind declares itself powerless? I do not want to believe this")

The next year in Zurich, seven Frenchmen attended, the good example had been followed by JACOD, JACQUES, LEMAITRE, REBATTU and VERNET

## FOUNDER MEMBERS

The photograph (fig 4) shows all who attended the first meeting The French and German members have already been mentioned In addition there was R BARANY from Uppsala (Sweden), the only otologist to receive the Nobel prize (for his investigations into the human vestibular organ) On one other occasion was the Nobel prize awarded for a study concerning the ear, the recipient was the physicist G VON BÉKÉSY — also a long time member of the Collegium Others attending from Sweden were BERGGREN and NYLEN (a pupil of HOLMGREN and successor to BÁRÁNY) There were no fewer than five Danes There were the wellknown clinicians BLEGVAD and LUND, there was MYGIND, whose merits lie especially in a comparative anatomical study of the vestibular labyrinth, there were BORRIES and THORNVAL, who had both carried out vestibular studies in the pigeon — the classic test animal of FLOURENS, BREUER and EWALD

England was represented by GRAY and WILKINSON, who have made important contributions to the anatomy of the ear and the physiology of hearing In addition there was TWEEDIE, popular from the start and the Collegium's first treasurer, and there was the eminent clinician CLEMINSON They also attended the Zurich meeting, where they were joined by FRASER, who had specialized in the microscopy of the ear, and VICTOR NEGUS, renowned for his studies of the larynx and paranasal sinuses, who was to become a notable figure in the Collegium These English colleagues were an important group, on whose attendance one could always count Apart from the classic English dignity they possessed considerable modesty, for in order to relieve the pressure of work on the general secretary they waived their right to receive circular letters in English It seems hardly credible today, but many of the official communications of the Collegium prior to World War II were written exclusively in French and German.

'LOCARNO SCIENTIFIQUE''

The attendance of the first meeting was highly satisfactory Of 58 physicians who had expressed themselves in favour of the suggestion made by BENJAMINS and DE KLEYN, 34 were present in Groningen They came from 10 different countries (9 from the Netherlands, 5 from Denmark and Germany, 4 from England, 3 from Sweden, 2 from France, Austria and Switzerland and 1 from Czecho Slovakia and Hungary) Italy was conspicuously absent on this first occasion, the contact, which later was to become so intensive, had apparently not yet been made Members from Finland, Norway and Spain did not attend, and Belgium had declined the invitation for the time being This was eight years after World War I

The attitude of the medical profession to war is a very special one The two world wars with their massed armies were made possible by the evolution of medicine But physicians must continue their duties and, in view of the Red Cross idea which does not discriminate between friend and foe, they must be above the course of events But war ruins so much in human interrelations that medical feelings, too, are subject to change In 1926 it was still difficult to arrange a meeting of people who had been enemies in the great war, at least a meeting on scientific, friendly terms The meeting in Groningen was probably among the first — if not the very first — of its kind, certainly it was the first meeting of otologists

Objections, as expected, arose chiefly from Western countries which had suffered most from the war Belgium and France Fortunately, two Frenchmen were nevertheless present There was brilliant GEORGES PORTMANN from Bordeaux, who from the start played an important role in the Collegium, there was MOURET from Montpellier – the investigator of the anatomy of the petrosal bone

From Germany came the wellknown clinician VOSS, his pupil GRAHE, ALBRECHT, WITTMAACK (whose original ideas and numerous animal experiments greatly inspired O R L research) and his pupil ECKERT MOBIUS – an authority on microscopy Of the Germans, VOSS from Frankfurt was the leading figure He was an exceedingly kind and good man, and kindness and goodness are important character traits in a physician The most remarkable man of the Germans was without any doubt the very scientific WITTMAACK He was often aggressive, his best pupil ECKERT MOBIUS called him once "a pike in a carp pond" But he was a good member of the Collegium in the first years During the banquet of the German O R L meeting in Hamburg he mounted suddenly on a chair and deeply moved he made the following speech "I am accustomed to the fact that I am not understood but now I insist to be heard and to be understood well For the first time after the war we have two distinguished Dutch guests, DE KLEYN and BENJAMINS We are very grateful that they are present here and we hope that this will be the beginning of a new era of scientific collaboration with the colleagues of the whole world " These words, which made a great impression, were spoken in May 1926, the Collegium started in October

None of those present will ever forget PORTMANN's dinner speech, it was

perhaps the best he ever made, and that means something! Recalling a pleasant conversation with Voss, he referred to an interview which shortly before had taken place between BRIAND and STRESEMANN This was the time of the rapprochement between France and Germany (l'histoire se repète), and the discussions between the two statesmen at Locarno had been productive Said PORTMANN in Groningen we have had "une Locarno scientifique"

ARISTIDE BRIAND was among the greatest politicians France ever produced The Paris memorial in honour of this statesman bears an inscription recalling one of his statements about war, which also has a bearing on medicine It reads „Les savants luttent chaque jour pour nous preserver de la tuberculose, du cancer et la guerre, l'horrible guerre serait le seul mal contre lequel l'humanité se declare rait impuissante? Je ne veux pas le croire "
("Every day, scientists fight to protect us from tuberculosis, from cancer, and should war — horrible war — be the only disease against which mankind declares itself powerless? I do not want to believe this")

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Fig 4 Foundation meeting of the Collegium Oto Rhino-Laryngologicum Amicitiae Sacrum, Groningen 1926

*First row left to right*
Barraud, Barany, Voss, Zwaardemaker, Burger, Mouret, Portmann, Gray, Marschik

*Second row*
Cleminson, Roorda, Blegvad, van Gilse, Berggren, Mygind, Albrecht, Thornval, Leidler, Grahe, Tweedie, Huizinga, Wittmaack, Nager, Botties, de Kleyn, Boonacker, Eckert Mobius, Versteegh, Szász, Lund, Wilkinson, Nylén, Precechtěl Benjamins

Switzerland was represented by NAGER from Zurich, who even in Groningen made himself indispensable as an interpreter For many years the custom (which has unfortunately disappeared for lack of time) was to present a brief summary of every paper and discussion in the two other languages Like Collegium member OPPIKOFER SR, NAGER was a pupil from the school of SIEBENMANN in Basel, famous for his studies of the microscopy of the petrosal bone Today, this great tradition is being continued in Zurich by ...

The ... famous Viennese school was represented by the renowned surgeon

MARSCHIK and by LEIDLER, an authority on neuro otology From Hungary came SZÁSZ, who carried out eminent animal experiments concerning the physiology of the labyrinth until his too early death From Prague came PŘECECHTĚL, an all round clinician who was to become a faithful member of the Collegium

Of the Dutchmen attending we must mention the international renowned BURGER, VAN GILSE (studies on the development of the paranasal sinuses), VERSTEEGH (one of the most brilliant co workers of MAGNUS and DE KLEYN), BOONACKER, ROORDA and HUIZINGA

## THE FIRST SCIENTIFIC MEETING

The international meeting in Groningen thus was a historic event, highlighted in this publication because we are concerned with history For the future of the Collegium, however, the result of the scientific discussions was of course more important The scientific meeting had been well prepared, and it was very successful Abstracts of the various communications had been circulated in advance This excellent custom has since been maintained, and was further improved when, upon a suggestion of VERNET it was decided to publish the abstracts in the three languages

In the two days of the meeting there were 16 papers, with 41 discussions — a well balanced programme exactly suited to the purpose of the meeting President ZWAARDEMAKER led the meeting with his customary kindness and erudition, and delivered the first communication on "The sense of smell" — a much neglected subject In the last few years there has been a turn for the better, but it was nevertheless not until 1963 that the next paper on the sense of smell was read in a meeting of the Collegium (by BOCCA and BATTISTON)

From the start there have been papers read on invitation (usually two), as a rule about personal investigations which had attracted special attention, reviews of topical subjects were occasionally presented In Groningen, Voss spoke about "Geburtstrauma und Gehororgan", at that time a new subject which later receded into the background when after World War II the emphasis was on intra-uterine injuries of the product of conception PORTMANN discussed "Recherches sur le sac endolymphatique Resultats et applications chirurgicales", this subject has lost none of its topical importance On the basis of a comparative anatomical study and animal experiments, it was maintained that MÉNIÈRE's disease is based on an "auricular glaucoma" Drainage of the saccus was recommended as therapy for certain patients This operation became an accepted procedure, which is still being used In this context it should be mentioned that, at the festive Groningen meeting 12 years later, HALLPIKE delivered an address on invitation on "Some recent work on intralabyrinthine pressure", in which he reported on the renowned microscopic studies with CAIRNS, demonstrating that labyrinthine hydrops does exist, at least in several cases

No fewer than 13 of the 16 papers read concerned the ear, while only 3 were devoted to other subjects As will be discussed later, most of the research done in O R L concerns this exceedingly important organ, nevertheless, however, this seemed a disproportion The auditory organ was the subject of papers read by GRAY and WILKINSON on "A restatement of the resonance theory of hearing" While the first travelling wave theory was formulated in the course of the previous century, its major corroboration was not presented until VON BÉKÉSY published his observations in 1928 Although many objections had already been made, the theory of HELMHOLTZ was still almost generally accepted in 1926 A major difficulty lay chiefly in the sharp localization on the highly damped basilar membrane In order to rescue the theory, GRAY and WILKINSON were the first to presume that a sharp localization resulted from maximal stimulation This

presumption became the starting point for subsequent theories It is still a great pleasure to read the discussions then conducted by BARANY, WITTMAACK and ZWAARDEMAKER

It is a conspicuous fact that no fewer than 10 of the 13 reports on the ear concerned the vestibular organ, while only 3 dealt with the hearing organ, which is so much more important to man This must be explained on the basis of the circumstances prevalent in the Netherlands at that time

## OTO-RHINO-LARYNGOLOGY IN THE NETHERLANDS ABOUT 1926

It was possible to organize the Groningen meeting because the Netherlands had been exempted from World War I, and because O R L in this country had attained a high level of excellence The top-ranking otologists of the world included a number of Dutch investigators out of proportion to the size of this country We mention ZWAARDEMAKER, BURGER, STRUYCKEN, BENJAMINS, QUIX, VAN GILSE, DE KLEYN and VERSTEEGH And scientists other than otologists, too, intensively concerned themselves with work concerning the ear This was chiefly a consequence of the activities of MAGNUS

In experiments on decerebrated animals, pharmacologist MAGNUS observed striking reactions of the limbs resulting from changes in the position of the head Through ZWAARDEMAKER's good offices, the so productive cooperation with DE KLEYN started in 1911 The latter designed the now classic operation for removal of the labyrinth in mammals For the first time, moreover, labyrinthine reflexes were distinguished from cervical reflexes There followed a careful analysis of the labyrinthine attitudinal and postural reflexes A more profound understanding of the otoliths became a necessity This led to the comparative anatomical studies of DE BURLET An investigator who confines himself exclusively to the peripheral labyrinth cannot hope ever to discover the complete truth which is the goal of all science The central nervous system is at least equally important in this respect Once it was found necessary to consider this in detail, the collaboration with surgeon RADEMAKER was begun Other collaborators of importance were VAN DER HOEVE, BIJLSMA, LE HEUX, DUSSER DE BARENNE and VERSTEEGH

In 1924, MAGNUS published his "Korperstellung" — a summary of the considerable body of work accomplished in the Utrecht pharmacological laboratory In the terms of purely vestibular physiology a new achievement of importance was an accurate description of the function of the otoliths certain reflexes were derived from the utriculus and the sacculus Another wellknown theory in those days was that of QUIX The question whether the adequate stimulus of the otolith was suspension according to MAGNUS or pressure according to QUIX was the subject of heated discussions at that time Both theories were in fact to be disproved In 1927 VERSTEEGH reported that it was possible to remove the sacculus in rabbits without affecting the reflexes ascribed to it Not until much later were VON HOLST and VON BÉKÉSY to demonstrate that the adequate stimulus in the labyrinth (and the same probably applies to all sensory spots) most likely arises

from shearing forces, thus they returned to the views previously held by BREUER The same had been demonstrated by STEINHAUSEN and ULRICH for the utriculus in pike, but these investigations attracted less attention than those concerning the crista

The influence of the school of MAGNUS and DE KLEYN was very considerable Until then, lower animals had been most frequently used in vestibular investigations, but now there was a marked increase in the number of centres doing experimental work on mammals This was promoted by the many investigators from abroad who came to work in their laboratory for varying periods of time Even if we confine ourselves to those visitors who were Collegium members, the list is impressive It includes BARÁNY, BERGGREN, CLEMINSON, GRAHE, LEIRI, LORENTE DE NO (the first member from Spain), LUND, MACNALLY, MEURMAN, MYGIND, NYLÉN, RUTTIN, TWEEDIE It is consequently understandable that 10 of the 16 papers read at the first meeting concerned the vestibular organ And something of this influence must have lingered, for papers on this subjects have been read at all meetings since, and an excellent symposium was presented under ARSLAN and DOHLMAN at the Padua meeting in 1960 Although as early as 1926 at a party during a Hamburg meeting it was already said that "the time had gradually come to stop "mit diesem Schwindel vom Labyrinth", considerable work on the subject is still being done, and will continue to be done in the foreseeable future

## OTO RHINO LARYNGOLOGY ABOUT 1926

We have reduced our discussions of O R L in the Netherlands to minimum length, mentioning only such facts as could elucidate the felicitous start of the Collegium Our review of O R L in general must even be more succinct The work on the vestibular organ done in the Netherlands, particularly in Utrecht and also in Groningen (with the pigeon as test animal), and which was to engage the intensive interest later of Amsterdam investigators under DE KLEYN and his successor JONGKEES, was started at a fortunate time Science, too, has its fashions", dependent on the repercussions of certain discoveries made at a given time With regard to the vestibular organ, FLOURENS was as far ahead of his time as MENDEL's theory of heredity was ahead of his days Even MÉNIERE had hardly attracted attention Vestibular physiology was given its start later, by GOLTZ Then the world was ready for the investigations of MACH, BREUER, CRUM BROWN EWALD, HÖGYES The endolymph flow as an adequate stimulus of the crista was generally accepted

Although investigations continued, a period of relative tranquility began shortly after But a few years prior to the publications of MAGNUS and DE KLEYN, emphasis was placed on the vestibular organ in studies by BARÁNY and many others, heralding the hayday of socalled otoneurology It should be borne in mind that the earlier authors were nearly all physiologists, or at least no otologists, whereas the publications on the crest of the new wave of vestibular investigation were for the most part written by otologists At the first wave which started after 1870 we may recall otology was still in its infancy

Modern otology started in Great Britain and Ireland, halfway down the previous century, with the Dublin investigator WILDE (OSCAR WILDE's father) as the great clinician, and TOYNBEE of London as the great scientist Both men were born in the year of the battle of Waterloo TOYNBEE was the first to give otology a more solid foundation by careful pathological anatomical examination of many otopathies One of his pupils was POLITZER But it was VON TROLTSCH of Wurzburg (inventor of the frontal mirror) who brought this approach which revolutionized both scientific otology and practice (more appropriate treatment) to the Continent Initially for the most part macroscopic but later also microscopic studies became almost entirely the realm of otologists They encountered numerous problems (acute and chronic otitis, cholesteatoma, defective hearing, deafness, otosclerosis, etc ), many of which are still among today's topics

In 1926, hearing was still determined in virtually the same way as half a century earlier, it was done chiefly with tuning forks, quantitatively and qualitatively, with the aid of the classic tests of WEBER, RINNE and GELLÉ World War I changed remarkably little in otology, whereas World War II was to bring great changes indeed But in 1926 the tempo of life was not yet so high as it has become in today's crazy world During World War I, the vacuum tube had been introduced for radiotelephony This was to be of importance to audiometry, and this was first realized in the USA The pitch range audiometer had been presented by BUNCH and DEAN as early as 1919, when in 1922 the first Western Electric audiometer (vacuum type) was introduced by FOWLER and WEGEL But in 1926 the audiometer was still unknown in Europe Otology goes west!

Very simple apparatus was also used in examination of the vestibular organ rotating chair and ear syringe Observation of the patient was the mainstay of these studies And in test animals, the consequences of certain operations on the labyrinth were studied At best there was some form of simple mechanical record ing of the nystagmus according to BARTELS Not without reason we mentioned the fact that the Nobel prize was twice awarded for research on the ear, BARANY in 1914 and VON BEKESY in 1961, Hungary can be very proud of these two excellent sons BARANY received the prize mainly for his work on caloric nystagmus, the only instrument he needed for this work was the ear syringe In the case of VON BÉKÉSY we are impressed by test arrangements for his brilliant experiments with a considerable body of apparatus Much had changed

In 1926 otology still largely confined itself to the treatment of acute and chronic otitis with numerous complications, which called for frequent and sometimes extensive surgery The operations on the nose and paranasal sinuses had for the [illegible]

that most human of all functions speech The field was greatly enlarged when endoscopy was introduced, which in 1926 was mainly used for removal of foreign bodies Endoscopy was almost exclusively carried out by laryngologists, but not much scientific work had been done on the subject, with few exceptions BRUNINGS from the KILLIAN school in Europe, and CHEVALIER JACKSON in the USA

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became larger and larger In view of the beautiful, interesting excursions, many members must have felt inclined to play truant and join the ladies It is a remarkable fact, however, and an indication of the members' scientific interest, that few have yielded to this temptation Joint excursions were later organized more frequently, for the afternoons And after congresses there were often organized visits to clinics and laboratories in the host country

Many friendships have been born within the Collegium, and they acted in favour of regular attendance One went to a meeting partly to see old friends It was like that before World War II, and it has become the same since It can even be observed, with considerable satisfaction, that Collegium friendships were continued across enemy lines during the war, and that human lives have been saved as a result

Perhaps this intensive personal contact in the Collegium has been more important, even in scientific terms, than the meetings themselves For, after all, the communications can be comfortably read at home after the congress, but then often lively discussions during and after meetings cannot be recaptured This intimate contact also gave us a much better understanding of the work done by others, and this was further improved by many visits to clinics and laboratories The resulting bonds of friendship have been very useful especially to the younger colleagues In later years in particular there have been intensive exchanges of assistants and ex pupils, and these were often made welcome in a special way

It can be described as fortunate, therefore, that the programme has from the start been a full day programme combining science and entertainment It has been repeatedly emphasized that entertainment was to be of a simple kind, avoiding [illegible]

a map of the city in advance, with instructions to carry this well visible for recognition at the station, where all were personally met There were two dinners The banquet (still without the ladies) was held on the first day, and a simpler meal was provided on the second day The respective costs were 9 shillings and 5 shillings (without wine) The simplicity of the fare did not in the least affect the gaiety of the occasion To the surprise of many colleagues, MYGIND managed to tempt WITTMAACK into joining him in an exuberant dance, perhaps these great experts on the labyrinth found this an attractive form of vestibular investigation

Much of the initial simplicity disappeared in later years, and we have known some grand receptions The Collegium gradually attained some fame, and this led to several inevitable official receptions, many of which were greatly appreciated It was our privilege to be received by the Pope (1950) and by the presidents of two Republics (Finland in 1951 and Ireland in 1958)

— *The first list of members* was published on 1st August 1927, just before the Zurich meeting There were 81 members from 15 countries, 5 are not yet mentioned The Collegium had already expanded across the Atlantic The two American members attending were DENCH from New York and G E SHAMBAUGH SR

from Chicago Belgium was fortunately also represented, by LEDOUX from Brussels, and members from Finland, Norway and Spain were also present Among the first members from those countries, mention must be made of LEIRI and MEURMAN SR from Helsinki, who became very active members and read papers on many occasions

Among the countries already mentioned, some have lent a special colour to the first Collegium years The Austrian membership, for example, included many famous names from the Viennese school In addition to LEIDLER and MARSCHIK there were ALEXANDER, BECK, BRUNNER, OTTO MAYER, NEUMANN, RUTTIN and later HAJEK Numerous colleagues all over the world have enjoyed their lectures. Perhaps we also liked to go to Vienna for its very special, attractive atmosphere, which is well defined in a saying dating back to about 1920, when Austria suffered so much from the war and from inflation "In Deutschland ist der Zustand schlecht aber nicht hoffnungslos, in Österreich ist der Zustand hoffnungslos aober gaor nicht schlecht" ("In Germany, conditions are bad but not hopeless, in Austria conditions are hopeless but by no means so bad") In Graz there was ZANGE, who later went to Jena, he was a fine clinician and an authority on labyrinthine microscopy ZANGE has also a great reputation as a teacher, amongst his pupils are f i WULLSTEIN and ZOLLNER

Of the English members we must mention BROWN KELLY from Edinburgh, whose efforts saved the Collegium from neglecting the oesophagus Of the French we mention BALDENWECK, with his intensive interest in the vestibular organ

There were three outstanding personalities from Scandinavian countries SCHMIEGELOW from Copenhagen, HOLMGREN from Stockholm and DOHLMAN from Uppsala (later professor in Lund), who did excellent experimental work on the vestibular labyrinth and also made early studies on allergy Those were the days of great personal authority exercised by a few prominent O R L specialists It is hardly conceivable today that a single person might exert such influence Examples are SCHMIEGELOW and HOLMGREN

SCHMIEGELOW was an all round clinician who presided the first international congress of O R L in Copenhagen in 1928 (all subsequent presidents were Collegium members), on that occasion he also presided the business meeting of the Collegium But at that time he was already approaching the conclusion of his career He came to a meeting of the Collegium in Copenhagen in 1948, when he was very old The meeting rose to a man when he entered, and this moment of homage has been unforgettable for all present

HOLMGREN was an excellent organizer with great merits for the cause of O R L He is sometimes called the father of the fenestration operation, although the first operations of this type were performed by our member JENKINS in London (1913) and later by BARANY He inspired our Collegium member SOURDILLE, who in his turn greatly influenced American colleagues And he brought BARANY and VON BEKESY to Sweden HOLMGREN had an instinct for what is important in science, BARANY is not the only one to owe a professorship to him Students like to exaggerate but sometimes formulate ideas in a charming way Many years ago, a Stock-

holm student paper published a cartoon depicting two statues — a small one and a big one — with two students in front of them One asks "Who are they and what did they do", to which the other one replies "The smaller one is Nobel Prize winner ROBERT BÁRÁNY He discovered the vestibular organ The bigger one is GUNNAR HOLMGREN He discovered BÁRÁNY"

Undoubted personalities from Germany were DENKER and BRUNINGS DENKER — a pupil of BEZOLD — was a wellknown clinician (DENKER and KAHLER's textbook) with considerable scientific talents which he displayed as a young otologist by independent work on the pathological anatomy of deafmutism and comparative anatomical studies on the organ of hearing BRUNINGS was one of the most original personalities in O R L, who introduced new ideas concerning the physiology of the organs of hearing and equilibrium, and who had great technical gifts (endoscopy) Remarkably, he differed from many other prominent colleagues in that he showed but little interest in the Collegium Regrettable for the Collegium and in particular also for BRUNINGS himself He attended only one meeting (in Frankfurt) and participated in only one discussion, about so-called hearing in fish, but this sole contribution still merits full attention

[illegible]

who was one of those eminent personalities promoting the [illegible]

### THE BUSINESS MEETINGS IN GRONINGEN AND ZURICH, AND THE RULES OF THE COLLEGIUM

In the early years the scientific meetings were held on two successive days, with a business meeting on the preceding evening, followed by an informal get-together Later, the business meeting was held at the end of one of the morning sessions

It is not only to pay homage to the memory of the two founders of the Collegium that we copied the bulk of their first circular in extenso This letter in fact formulates principles which have determined the structure of the Collegium Five points from this letter constitute the basis of the rules of the Collegium as discussed in Groningen and formulated in 12 articles at the second meeting in Zurich

1 Only scientific subjects are to be dealt with, whereas purely clinical subjects are to be avoided This is no discrimination against clinical medicine, the vast majority of our members, after all, also have a certain reputation as practitioners Particularly when great new clinical possibilities were introduced after World War II, it has sometimes been difficult to keep the enthousiastic clinicians within the rules

2 The number of members is to be limited to a maximum of 10 from each country This never caused any real difficulty It might have been expected that larger countries would claim the right to send more members than smaller nations, in fact this has been discussed once or twice at business meetings But it was immediately felt that a decision about proportionate representation might readily lead to some form of chauvinism Apart from that, a small country can very well be

great in science In fact the attendance of members from some of the so called smaller countries was often much better than that of members from some larger countries

3 More time is to be given to communications Regrettably, the 30 minutes originally set aside per paper had to be gradually reduced to 15 minutes, as the number of contributions increased Only papers on invitation are granted more time, and a reasonable period is always set aside for discussions

4 The meeting languages are English, French and German in accordance with international-congress traditions The Italians, after joining in 1927, suggested that Italian be recognized as the fourth official language This was rejected because only a small percentage of members had any Italian and because acceptance, it was feared, might pave the way towards chauvinistic suggestions to accept a fifth language, and a sixth etc which would end up in a truly Babylonian confusion It was deeply regretted that the Italians then left the Collegium In 1932, fortunately, BILANCIONI, BRUNETTI SR and TORRIGIANI returned

5 If the plan was successful, a meeting was to be held every year in one of the participating countries Well, success was obvious and meetings were held every year, with the South and the North of Europe as alternate locations Of course the years of World War II were exceptions It was decided that during the year of an international congress, only a business meeting was to be held in the same place as the congress It was emphasized from the start that the Collegium must in no way be regarded as a rival of the international congresses The solution suggested above would in fact be beneficial to the international congresses It has been supposed that Collegiums might also be founded elsewhere, but this has not happened

A number of other articles concern the executive Committee and the members The Committee originally had 5 members, of whom the general secretary and the treasurer were re eligible after a 3 year period (the complete trust which members showed in these officers made them virtually permanent Committee members) The president vice president and a member were to be elected every year (the former two from the country where the meeting was to be held) After the war the member was replaced by a permanent second secretary in charge especially of proceedings Because of the intimate relations with Acta Oto Laryngologica, one of its editors was elected For many years this post was filled by SKOOG, who succeeded HOLMGREN in Stockholm and continued his great tradition in O R L He abdicated in 1963 his successor HAMBERGER became a member of the Bureau The important work in collaboration with Acta was continued by FRENCKNER, who had already replaced SKOOG in an excellent way during the last years In London (1954), a counsellor was added to the permanent Committee (GEORGES PORTMANN)

Members become corresponding members when they reach the age of 70 (after the war this has been changed to "honorary members") Under circumstances (resigning from practice, etc ), members can become honorary members before they are 70 making place for younger colleagues Few have so far availed themselves of this possibility

The election of new members is primarily a matter for the country concerned,

where members decide on a candidate by a majority vote When a candidate is accepted, the Committee is informed, which presents the candidate to the meeting With a view to stricter selection it was suggested even in Frankfurt (1930) that the candidate send reprints of his scientific publications to the committee It was so decided Since 1961 these publicatons have been first submitted to three members (DOHLMAN, LINDSAY and NEGUS, succeeded in 1964 by FRENZEL, MACNALLY and HUIZINGA)

It was decided that the subscription for the coming year shall be fixed at each meeting For years this has been 10 shillings, but after World War II there has been a rise to 3 pounds The prosperity of the Collegium has been largely dependent on work behind the scenes, certainly also on the excellent financial management by the treasurers TWEEDIE, NEGUS and RUEDI

It was decided that manuscripts of papers had to be handed in ready for printing during the meeting or within 14 days The proceedings have been published from the start in Acta Oto-Laryngologica, with only three exceptions The proceedings of the first two special meetings after the war appeared in Acta Oto-Rhino-Laryngologica Belgica, and those of Rome (1950) in Revue de Laryngologie, Otologie et Rhinologie Today we have 24 proceedings of 20 ordinary and 4 special meetings It is good to see that so much attention has been paid to this important aspect Whatever the Collegium's future may be, these proceedings will always bear witness to the importance of its activities

### THE MEETING IN ZURICH IN SEPTEMBER 1927

Another neutral country was the obvious choice for the second meeting The presence in Groningen of NAGER, with his great authority also on the international level, made it easy to choose Zurich There were 27 papers with 71 discussions, the ratio being slightly more favourable than in Groningen The programme was fortunately no longer so one sided, 20 papers still concerned the ear, it is true, but only 4 of these discussed the vestibular apparatus, 9 concerned acoustics, 7 the middle ear or labyrinth in general There were two communications on invitation, by WITTMAACK and MOURET Both were originally to speak on pneumatization, but this intention was changed when this was found to be the subject of one of the reports of the 1928 international congress in Copenhagen MOURET discussed "Systematization of the mastoid and of mastoiditis, mastoidectomy' in a paper unmistakably of a clinical nature, but based on very careful anatomical studies It corresponded well with the views of the Viennese and the German school (NEUMANN had paradoxically stated "I do the radical operation conservatively, and the mastoid operation radically")

WITTMAACK discussed The functional importance of the organ of CORTI and the demyelinated nerve endings for the act of hearing", a paper read by BARANY was on similar lines, and both papers provoked exhaustive discussions of hearing theories A remarkable feature was WITTMAACK's view on hearing without organ of CORTI by differences in hydrostatic pressure" on the delicate demyelinated nerve endings Another remarkable paper was that by Voss and MULWERT on the

improvement of chronic defective hearing by ultrasonic treatment — now exclusively used for destructive purposes HERZOG from Innsbruck discussed hearing in Cetacea, referring to BOENNINGHAUS' wellknown anatomical studies Since PETRUS CAMPER, much has been written on the hearing organ and the absence or presence of hearing in Cetacea From experiments since made with dolphins we know with certainty that they do hear, and hear well, but not in the way imagined by HERZOG At the time it was unknown that precisely the perception of ultrasonic waves was so important An address by NAGER on "The ear and endemic cretinism" with a comprehensive demonstration of microphotographs from the Zurich clinic's large collection, attracted considerable attention No fewer than 9 members wished to participate in discussion

NEGUS, who was later to read many papers on comparative anatomy, discussed "Observations on the evolution of man from the evidence of the larynx" MARSCHIK presented the first film to be shown in the Collegium It concerned a patient treated by pharyngeal resection and hemilaryngectomy and showed the motions of the remaining laryngeal half in respiration, swallowing, speech, etc There were two papers on the anatomy and physiology of the oesophagus (by VAN GILSE and by JACQUES and ROUSSEAUX)

In Zurich, all members from abroad were accomodated in the same hotel — a good custom which before World War II became almost traditional and which greatly promoted the "Amicitiae Sacrum" We were greatly spoiled by our Swiss colleagues, who gave us a banquet in the Grand Hotel Dolder Nobody will ever forget NAGER's way of announcing some yodlers Some people had reported who used the voice in an extraordinary manner, and these laryngeal patients were to be demonstrated The yodling charmed us all, as usual, and is an interesting topic from a point of view of laryngeal physiology and especially of vocal technique As a good pupil of this school RUEDI was to treat us to yodling once again in this same place, 25 years later (in 1952)

### THE BUSINESS MEETING IN COPENHAGEN (1928)

introduced a new feature touching upon the tragic side of life For the first time, president SCHMIEGELOW had to commemorate two members who had died in the course of the preceding year MOURET and BECK Both had been active participants in the Zurich meeting They were the two members who opened the ranks of the many who have followed them since The list includes over 120 names at present, and these names will arouse emotions of melancholy and respect in the members still alive Melancholy because so many good friends are no longer with us, and respect because these names stand for a good part of the history of O R L in this century The members are frequently confronted by these names, which in every new members' list are presented under the heading "Tabula eorum qui supremum diem obierunt" At each meeting the list, veiled, is displayed on one of the walls of the meeting room

Life goes on, and new members were welcomed in Copenhagen Many came from Spain including CASADESUS and HINOJAR, who were to become regular

attendants, like FAIREN who had joined us in 1927 From Hungary also came new members, notably president to be REJTÖ and KELEMEN, a faithful and versatile member who has read many papers Another versatile colleague was ŠERCER from Zagreb — the first member from Yugoslavia After the war this country was to be eminently represented by GUŠIC and PODVINEC ŠERCER read many papers on a wide variety of subjects Another important newcomer was TERRACOL, at the time from Strassbourg but later professor in Montpellier, who also became a faithful member with many interests It is a tragic development that with the enormous increasing specialization in the past few years we find an inevitable diminution of the ranks of many sided investigators such as KELEMEN, ŠERCER and TERRACOL, and the race of truly versatile O R L specialists may well be extinct soon

It was decided to invite Russian colleagues Moscow professor WOJATSCHEK was asked to appoint candidates, and four Russians joined the Collegium the next year KOMPANEJETZ, MALIUTIN, UNDRITZ and WOJATSCHEK, who have read a number of good papers It is still regretted that contacts have not been immediately resumed after World War II

## LONDON JULY 1929

The London meeting under the presidency of A A GRAY was a very special event To begin with, it was the best attended meeting prior to World War II, because no fewer than 64 of the 104 members attended Proportionally this was the alltime top attendance London seems an attractive place for an international meeting In the western world, moreover, the economic and political circumstances were favourable, or at least showed nothing of the murkiness which was to characterize many later years The participants are shown in the photograph (fig 5)

Another noteworthy feature was the presence, for the first time, of 7 American colleagues, including 2 guests Since the foundation of the Collegium, a few guests have been invited at each meeting, the invitations require the approval of the executive committee There were 4 members from the USA GUILD from Baltimore (a faithful attendant later), POHLMAN from Buffalo, SHAMBAUGH SR and WILSON from Chicago, and there was one member from Canada BIRKETT Thus the Collegium witnessed the first scientific contact across the Atlantic which was to be repeated many times and to yield valuable results WILSON, an authority on labyrinthine microscopy, discussed "The utriculo endolymphatic valve" POHLMAN and SHAMBAUGH read papers on acoustics which — against the background of those days — were exceedingly important The former discussed "Correlations in the sensitivity to air and bone transmitted sounds, with a note on the negative pressure treatment" Negative pressure in the meatus has not been widely accepted in the treatment of deafness (as it has in diagnosis VAN DISHOECK) A more important fact was that the majority of members made their first acquaintance with the audiometer This was exclusively the subject of SHAMBAUGH's communi



Fig 5 *London July 1929*

*First row left to right* Dohlman, Birkett, Tweedie, Nager, Fraser *Second row* Negus, Ledoux, Vernet, Benjamins, Burger, Portmann, Gray, Holmgren, Shambaugh, Wittmaack, Moller, Marschik, Barraud, Boonacker. *Third row* Lorenz, Berggren, Albrecht, Voss, Ruttin, Barbey, Tapia, Wiskovsky, Přecechtěl, Ninger, Ulrich, Mayer, Logan Turner, Gording, Denker, Roch, Jenkins, Huizinga, Wilkinson *Fourth row* Borries, van Gilse, Pohlman, Leidler, Fairén, Hinojar, Schlittler, Leegaard, Portela *Fifth row:* Kelemen, Guild, Gordon Wilson, Wodak, Šercer, Luscher, de Juan, Cleminson, Grahe, Kistler, Versteegh, Garcia Hormaeche, de Kleyn, Thornval, Nylén.

cation on invitation, entitled "Evaluation of the usefulness of the Western Electric audiometer in solving clinical problems for the practising otologist". SHAMBAUGH presented a very critical review, for various purposes such as study of the low frequencies, tuning-forks were still preferred We know that the introduction of the audiometer met considerable resistance on the part of otologists who, after all, had for more than a century been using tuning forks in determining hearing As has been pointed out it is remarkable that numerous American otologists were already using the audiometer in the twenties, whereas its introduction in Europe did not come until the thirties

The second communication on invitation was FRASER's paper on "Non experimental labyrinthitis in animals" WITTMAACK demonstrated numerous microphotographs concerning "so-called experimental otosclerosis in hens" There followed a rather heated discussion with OTTO MAYER, which was continued the next year in Frankfurt, when MAYER presented his views on the pathogenesis of otosclerosis On the basis of his experiments with fowls, WITTMAACK believed that venous congestion was the cause According to MAYER, the otosclerotic foci were to be regarded as masses of callus resulting from spontaneous fractures said to be common in the labyrinthine capsule

The London meeting was held in the hallowed hall of the Royal College of Surgeons, where the business meeting also took place It was decided that Polish colleagues were to be invited to membership They were LASKIEWICZ, LUBLINER, SREBRNY, SZMURLO and ZALEWSKI A somewhat naive decision was that every member was to learn one foreign language a year, which would soon obviate the need to engage translators After conclusion of the meeting we went with the ladies to see the famous Harvey film Even in a city the size of London did the company succeed in remaining together The final evening was devoted to a very special "at home" with the CLEMINSONS, in whose garden an enormous tent had been converted into a perfect ballroom

## FRANKFURT SEPTEMBER 1930

The Frankfurt meeting under the presidency of Voss, laid even greater emphasis on companionship The meeting had been extended to cover 3 days, still with the business meeting on the preceding evening The afternoons, it was planned, were to be devoted to excursions But there were no fewer than 33 papers to be read (the maximum pre war number), and one afternoon was consequently devoted to science On the remaining two afternoons, however, members were motored through the autumnal splendour of the Taunus mountains One afternoon we had lunch in the Konigstein Kurhaus (3 Marks, including beverages!), and the next afternoon we were the guests of the resort town of Homburg As in Zurich and London, we were all accommodated in the same hotel, the renowned "Frankfurter Hof"

There was only one communication on invitation, TERRACOL being unavoidably detained DENKER spoke on Hearing ability in fish" For many centuries, absence or presence of hearing in fish has been a question of importance to fishermen,

particularly through VON FRISCH's work, it had in recent years attracted the attention of scientific investigators The question remained whether one could really refer to "hearing" in fish A film was also shown, even without it, however, DENKER's presentation was sufficiently demonstrative to attract several ladies (this is an exception in the Collegium, although the fair sex often shows an interest in the presidential address)

An interesting contribution was a paper by GUILD, CROWE, BUNCH and POLVOGT on various microscopic findings in defective hearing, illustrated by numerous photographs and audiograms The first papers by Russian colleagues were also presented at this meeting (MALIUTIN and WOJATSCHEK) MALIUTIN discussed stroboscopic findings obtained in singers, stating that the left vocal cord remained non vibrating in 20% of these normal individuals One would hazard a guess that imperfections of apparatus in those days must have played a role, for non-vibration of one vocal cord is today regarded as a very important symptom In 1930, stroboscopy was used by regrettable few laryngologists BERGGREN presented an excellent analysis of a case history with bouts of dizziness, which was the subject of lively discussion This female patient was originally DE KLEYN's, GRAY examined the petrosal bones and BERGGREN the CNS — an excellent example of teamwork in the Collegium

BROWN KELLY read a paper on "Congenital stenosis of the oesophagus with diaphragmatic hernia of the stomach" He was the first to describe this condition, which has since attracted considerable attention and has been the subject of many reports in the literature Partly because of the pioneer work done by laryngologists, it is no longer possible to refer to the "oesophagus that unknown organ" (BELINOFF) As many as 8 colleagues participated in a discussion provoked by KELEMEN's paper on growth inhibition of tonsillar extract on tissue cultures The function of the internal secretion of the tonsils has been a much discussed subject, today our views are entirely different ŠERCER read a remarkable paper on respiratory function, about the difference between nasal and oral breathing

The Frankfurt meeting was a great success, both for science and for international friendship Moving was a short poem printed at the bottom of the usual group photograph "Ein jedes Band das noch so leise, die Geister aneinander reiht, wirkt fort in seiner stillen Weise, in unberechenbare Zeit" (*"Even the slenderest band that links minds together, persists in its quiet way incalculable far into the future"*) — In the business meeting HOLMGREN, PŘECECHTĚL and ZWAARDEMAKER were reported absent due to illness Telegrams of sympathy were sent, as was to become tradition ZWAARDEMAKER died soon after, he was the first president to pass away, the other two members made a good recovery

## THE BORDEAUX MEETINGS

Twice we went to Bordeaux, in July 1931 and in September 1956 Both meetings were presided by the ever dynamic PORTMANN Both PORTMANN himself and the Collegium must have considered it a privilege to have the two meetings — 25 years apart — under the same presidency The two Zurich meetings, too, were

25 years apart (1927 and 1952), but the second meeting was presided by RUEDI, although NAGER was still alive and well (he died in 1959) and he was proud of the success of the meeting and of his pupil Groningen and London each also had two meetings, but in both places the president of the first meeting was not alive to attend the second

In 1931 LEDOUX presented a theoretical review of "Radiosensitivity and karyoclastics", he was an authority on this subject The relation between radiosensitivity and breakdown of cell nuclei had been known since 1903, but had become a focus of interest chiefly due to the work of Paris investigators such as REGAUD and COUTARD in recent years The action of karyoclastics has much in common with the effect of X rays, but their practical application has had little success, they have been ousted out by the cytostatic drugs, with an entirely different mechanism of action The second communication on invitation was GUILD's paper on "Correlations of histologic observations and the acuity of hearing" It was found that it is often exceedingly difficult to evaluate this acuity on the basis of microscopic findings only In particular the number of ganglion cells in the spiral ganglia was less reliable as a source of information than had been suggested at the previous meeting in Frankfurt GUILD's paper was an exhaustive review with justified criticism of the determination of hearing, both with tuning forks and with the audiometer (standardization is necessary), for the first time, mention was made of the 1930 report on investigations made by WEVER and BRAY

The photograph (fig 6) shows all those present We immediately see that the company is much smaller than that in London, in fact the attendance was the lowest ever 33 members Certainly this had nothing to do either with the host or with Bordeaux But at this time Europe had received the full impact of the economic slump This was why one of the articles of the Collegium's rules, not previously mentioned, was never applied It had been decided in Groningen that any member absent at four successive meetings would lose his membership Recognizing the fact that for some members it was at that time difficult to travel far, the article was not enforced Later, political circumstances made it impossible to do anything but ignore the article in question

Those absent from the Bordeaux meeting missed much, both scientifically and in terms of entertainment All participants, both in 1931 and in 1956, have the fondest memories of the cordial welcome they received at MR and MRS PORTMANN's beautiful country house "L'Abbaye de Bonlieu" Madame PORTMANN has always been a faithful supporter of the Collegium She is a daughter of the famous MOURE, the first professor of O R L in France We all dined at the house, and afterwards the people of St Eulalie, the village of which PORTMANN was burgomaster, serenaded us with hunting horns ("le son du cor") And the wine! We saw ancient cellars and drank Chateau Yquem On the last evening, at the banquet in Saint Emilion (a Girondist stronghold), we were treated to no fewer than 18 wines "grands crus" This had its inevitable consequences There was a small party who had noticed that Saint Emilion was situated to the East of Bordeaux, that is on the way home After dinner they decided to motor home They departed and travelled all night In the morning they reached Bordeaux



Fig. 6. *Bordeaux July 1931*

*First row left to right* Thornval, Barraud, Guild, Gray, Ledoux, Portmann, Benjamins, Tweedie, de Kleyn, Přecechtěl, Nager.
*Second row:* Szmurlo, Rejto, Roch, Rebattu, Wiskovský, Fatrén, Casadesús, Freystadtl, Leiri, Day, Negus, Ulrich.
*Third row.* Boonacker, Vernet, Huizinga, Kistler, Jacod, Barbey, Lüscher, Leegaard, Retrouvey, Roorda.

In 1956 we renewed our acquaintance with the wine, devoting three afternoons to a tour of famous wine districts Several members had the privilege of being installed as honorary members in the jurades PORTMANN is Senator for a wine department, and he let us know it!

In 1932, during *the Madrid international congress*, there was only a business meeting under the presidency of TAPIA, wellknown laryngeal surgeon

PRAGUE SEPTEMBER 1933

We were in Prague with PŘECECHTEL as president and AMERSBACH as vice president, at that time there was also a German University in Prague We saw the beautiful historical buildings of the old city, and heard fine Czechoslovakian folksongs But science was the main thing CROWE reported on investigations he had made over a 3 year period with HUGHSON and HOWE according to WEVER and BRAY There had been considerable doubt as to the significance of the phenomenon, but on the basis of some 700 experiments the conclusion was formed that it is an excellent yardstick of the quality of hearing The second paper on invitation was presented by NEUMANN, discussing "The pathology and treatment of otogenic meningitis" This seemed a clinical subject par excellence NEUMANN was above all an eminent clinican, but he demonstrated how such a subject could be treated in a scientific manner Re reading this paper one cannot help thinking of the great change brought about by the introduction of antibiotics, at that time, a 37% cure rate in purulent meningitis by acute otitis was a very felicitous result

TAIT from Montreal read an interesting paper on "Evolution of vertebrate voice" Especially in view of observations on frogs and comparative anatomical studies he concluded that the swimming-bladder in fish must have originally been intended for the production of sound His views contrasted with the more or less generally accepted theory that voice is a secondary function arising from the respiratory organs Of course a discussion with NEGUS developed The physiologist TAIT was known to us especially for his collaboration with MACNALLY on the vestibular organ in frogs The two investigators presented a paper on this subject, on the basis of fine experimental work they formed the conclusion that the utriculus in frogs has a compensatory function with the contralateral vertical semicircular canals

STOCKHOLM SEPTEMBER 1934

At this meeting presided by HOLMGREN, two especially fine papers on invitation were presented by recognized authorities in their fields The anatomist DE BURLET discussed "Comparative anatomical data on endolymphatic and perilymphatic sensulae of the labyrinth", and NEGUS dealt with "The mechanism of phonation", as expected of this speaker, his paper was based on a comprehensive comparative anatomical study of the larynx

Another outstanding paper was that presented by DUEL from New York on "Surgical treatment of facial nerve paralysis" With BALLANCE in London, he had

carried out experiments in monkeys and cats to study the use of grafts, which were later to be applied to patients DUEL was among the first who, in view of animal experiments, began to slit the sheath of the facial nerve in Bell's palsy

On the way home, some members visited the clinics in Lund and Copenhagen in both cities we were given a very cordial welcome by our Scandinavian friends

### ENTERTAINMENT

Of course this must remain a secondary matter, but a member of the Collegium has the privilege of meeting some hosts who organize events which he is unlikely ever to forget One of these was a reception in the Stockholm City Hall, after the business meeting we were joined by the ladies and — preceded by a choir of singing students — proceeded in a stately procession to the banqueting hall Then there was the old Royal Theatre Drottningholm, made ready for a reception after a century of slumber NYLEN's children danced a menuet for us Another highlight was the banquet at the Budapest Hotel Hungaria, after which we were treated to Hungarian folksongs The famous violinist MAGYARI IMRE "played into the ear" of the lady singer, and for a long time we breathlessly listened to perhaps the best gypsy music ever played. And in Venice we were at the Lido Excelsior, and a special boat brought us along the romantic canals to the meetings and to the banquet at the Royal Danieli, where we were serenaded After the war, too, there were many beautiful and interesting events, ranging from a reception at the Capitol in Rome to folkdances in Zagreb, Athens and Edinburgh, ballet in Vienna, horses in Ireland, wine in Bordeaux, music in Wurzburg, and many other treats

### BUDAPEST SEPTEMBER 1935

President was REJTO, who delivered an address on the work of pathologist HOGYES, whose interest in the labyrinth, like MAGNUS' interest, was based on a coincidence He studied the influence of rapid rotation on the rectal temperature in rabbits, and happened to notice post rotatory nystagmus From 1879 to 1886 he presented 16 publications on the labyrinth, but exclusively in Hungarian Only in 1913, through SUGAR's German translations, did the world receive information about the important findings he obtained Among other things, HOGYES was the first to describe rotation nystagmus in man

On invitation, WOJATSCHEK discussed "Clinical determination of the function of the otoliths", while FARKAS spoke about "hearing" in fish, surprising his audience by attaching importance to the cristae Mention must also be made of a paper by DE KLEYN and VERSTEEGH on human reactions on the tilting table according to RADEMAKER and GARCIN, it corroborated the excellent study by MACNALLY and TAIT in frogs, about which a film was shown

### VENICE SEPTEMBER 1937

The meeting was presided by BRUNETTI, who honoured the memory of two especially meritorious members deceased in the course of 1936 GRAY

and TWEEDIE, who had never missed a meeting GRAY epitomized the exact scientific investigator and was a very kind man, as was TWEEDIE, whose knowledge of the international literature was amazing and who had done so much to ensure the friendly spirit in the Collegium

Communications on invitation were NAGER's paper on "The osseous pathology of the labyrinthine capsule", with again an impressive array of microphotographs, and TERRACOL's review of "The sympathetic innervation of the larynx" — a difficult subject indeed Other outstanding papers were presented by ECKERT MOBIUS, discussing a comparative-anatomical study of pneumatization with special reference to pneumatization in birds, by HALLPIKE, discussing the WEVER and BRAY effect under microscopic control, by ŠERCER on the pathogenesis of septum deviations, and by VAN GILSE on the pathogenesis of exostoses in the external meatus

The business meeting elected NEGUS treasurer, he proved to be as devoted as TWEEDIE, and upheld a certain tradition TWEEDIE liked to give an after dinner speech in French And we have since continued to enjoy many examples of "oune petite histoire", e g about "le chapeau brun" or about "le jeun messiou qui a perdou sa montre et qui la cherchait a oune place avec plus de loumière" In fact we have been fortunate in having been treated to many very good after dinner speeches at banquets

## GRONINGEN JULY 1938

This jubilee meeting was held under the presidency of DE KLEYN, deputizing for BURGER who was absent due to private circumstances The attendance was below expectation, there were 51 members (the membership then being 127) Perhaps the impending disaster of another world war already made itself felt The intention was to give this meeting — the tenth — a festive colouring, and this was why DE KLEYN opened the meeting with an address which briefly reviewed the Collegium's 12 year history

Until then, a presidential address had been unknown in the Collegium, partly also because the business meeting was still being held on the preceding evening Very often, not all members had arrived at that time, and the custom was consequently dropped after the war The day before the first meeting is now used as registration day, often with a reception in the evening hours At the business meeting, of course, some words of welcome were always spoken, but they were nothing like a presidential address The next address was to be delivered 12 years later, by FERRERI in Rome (1950)

We have already mentioned HALLPIKE's Groningen communication, the second paper on invitation, by HOLMGREN and NAGER, attracted considerable attention The former reported on the fenestration operation with special reference to closure of the fistula NAGER demonstrated the microscopic features in the monkeys on which HOLMGREN had operated, taking various measures to prevent closure of the fistula Even in the first postwar years this was the crucial problem, until ROSEN's stapes mobilization presented new problems




Fig 7 *Groningen July 1938*

*First row left to right* Dohlman, Birkett, Jacques, Holmgren, Berggren, Benjamins, de Kleyn, Negus, Nager, von Eicken, Brunetti, Brown Kelly *Second row* Skoog, Roorda, Cleminson, Kelemen, Luscher, Thornval, Meurman, Mygind, Podestà, Boonacker, Zange, Barbey, Roch, Ferreri, Wodak, Ulrich, van Gilse, Lionel Colledge, Nylén *Third row* Soueddle, von Gyergyay, Rejto, Vernet, Ledoux, Oppikofer, Torrigiani, Cambrelin, Přecechtěl, Chéridjian, Leegaard, Mollison, Sidney Scott, Blegvad *Fourth row* de Burlet, Versteegh, Huizinga, Frenckner

As a matter of course, entertainment had been given special attention It was customary, on one evening, to have separate dinners for the ladies and the gentle men, this was often highly successful, but the climate in Groningen was such that the company remained together There was much local colour We were serenaded by 200 children with castanets, we saw the roses of the castle of Menkema, and the stone graves in the province of Drenthe We had a lunch (a so-called "coffee table") with waitresses in folklore costumes, and enormous coffee pots on the help yourself principle It was great fun for nearly everyone except a few Frenchmen They had ordered Bordeaux wine and "it had not the right temperature" So good a country is „la douce France" that some French wish to live just like in France when they are abroad The banquet on the last evening was the highlight of the occasion A home made mixed grill was offered CHERIDJIAN from Geneva sang for us, accompanied by his wife, the 'Collegium-singers" gave a recital, and the "Collegium-springers" (fig 8) presented a wooden shoe dance (this seems to be expected in Holland), the performers were members' children with some little friends BENJAMINS had formed his own orchestra and played an invigorating march



Fig 8 The Collegium springers with their producer
First couple Marken second Volendam third Zeeland fourth Groningen
So typically Dutch!

## WAR

The 11th meeting was to be held in Brussels from 7th 10th September 1939, under the presidency of LEDOUX The programme had already been sent to the members and, as usual, promised much Communications on invitation were expected of VON BÉKÉSY ("Measurements of bone conduction hearing") and SOURDILLE who was to discuss "Tympanolabyrinthopexy a possible solution in the surgical treatment of otosclerosis" A provisional solution was soon to be introduced by LEMPERT in the USA, but the groundwork was done in Europe

On 1st September 1939, BENJAMINS circulated the following letter "Since the present international conditions threaten the success of the meeting intended, this will be postponed to a later date As soon as a decision in this respect is made, members will be informed"

Such members as received this letter, did so when the mad war had already started This letter was BENJAMINS' last service to the Collegium He died in February 1940, and the deputy secretary of course informed all members of this fact The loss was felt deeply when the Collegium resumed its activities after the war, as was the absence of MRS BENJAMINS, who had been with her husband at all meetings As MRS DE KLEYN would later, she always retained a deep interest in the further development of the Collegium, and both have appreciated it that many of the older members have evinced lasting feelings

The proceedings of the Groningen meeting were published only two years later in Acta Oto-Laryngologica, at that time the Netherlands were already occupied territory The Collegium started its period of hibernation with a membership of 132, from 26 countries But during and immediately after the war, numerous expressions of sympathy were received As early as 1946, letters came from NAGER and HOLMGREN with an invitation for a meeting in their country, but at the time this was still impossible

### POSTWAR REVIVAL OF THE COLLEGIUM

When restoration of international contacts became possible, we found that president LEDOUX was seriously ill, he died soon after The vice-president of the 11th meeting was HICGUET — a man with a wide ranging interest, especially in the ear, he introduced the term copho-surgery for the new hearing improving operations and was a good teacher to our members HENNEBERT JR and VAN EYCK, among others He and his friend CAMBRELIN took the initiative for the next meeting A preliminary discussion was arranged in Brussels in September 1946 between HICGUET, CAMBRELIN, NEGUS, VAN GILSE and HUIZINGA Of course the relations between colleagues from the various countries was again a serious problem Reluctance between former enemies is a consequence of war particularly of one that had caused so much human sorrow as World War II The result was that the Germans were for the time being not invited, and it was decided that an *extraordinary* meeting should be held in Brussels in September 1947 The organization of such a meeting still offered considerable difficulties, but

these were overcome in a pleasant collaboration with president HUGUET and vice president CAMBRELIN A need to see one another again was deeply felt, and the great changes which had occurred in the field of O R L called for careful discussion

## BRUSSELS SEPTEMBER 1947

There were only 33 honorary members and members, the ranks of the "old guard" had been severely thinned during the 9 years since the previous meeting in Groningen No fewer than 21 members had died, including such faithful Collegium members as BROWN KELLY, CLEMINSON, RUTTIN, former president REJTO, SZASZ, TORRIGIANI and ULRICH Yet the meeting had a good attendance for HUGUET had invited many guests There were in fact so many guests that the secretary admonished us not to exaggerate this in future lest the intimate friendliness of the Collegium be lost On the other hand, these invitations had good results in that some of the guests were soon to be good members of the Collegium, we mention AUBRY, BRUNETTI Jr, CAWTHORNE, SIMSON HALL, FOWLER Jr, SHAMBAUGH Jr and TATO, all of whom have read several papers since

As always, papers on the same subjects were arranged in groups In Brussels there were four main categories 1 *Audiometry* HALLPIKE and DIX reported on their widely renowned method of investigation for use in children ("peep-show"), VAN DISHOECK reported on his audiometer with constant intensity at varying frequency, with which dips are easily detected, HUIZING was one of the first in Europe to speak on recruitment — the important symptom first described by FOWLER SR 2 *The vestibular apparatus* in this category we saw a film by FOWLER JR on the action of streptomycin 3 *Fenestration* with papers read by such authorities as HOLMGREN, SOURDILLE and SHAMBAUGH JR 4 *Allergy* with an introduction by CAMBRELIN, followed by no fewer than 6 papers, including those of HERBERTS and HLAVACĚK on experiments in guinea pigs It is regrettable that BENJAMINS was no longer there to witness such interest in a subject on which he had reported a few times in the Collegium before the war

We were given an impressive reception at the beautiful city hall, where we were addressed by burgomaster our colleague VAN DE MEULEBROECK, known for his dignity of behaviour during the occupation There was an unusually attractive excursion to Bruges, where MR and MRS EEMAN made us welcome

While the Collegium had fortunately resumed its activities in Brussels, it had not yet completely started again The meeting at Brussels was an extraordinary meeting and three more such meetings were to be held In September 1948 we went to Copenhagen with BLEGVAD presiding, we enjoyed the proverbial hospitality of our Scandinavian friends The first evening there was a very animated open house at the home of the president and Mrs BLEGVAD, another evening all the members were invited to dinner parties in the various homes of our Danish friends There were no less than 69 members representing 18 countries From the 26 papers there were 21 on the ear and only 5 on other subjects But

some new topics were discussed, as by VAN EGMOND on "the BARANY test compared with cupulometry" about his wellknown work with GROEN and JONGKEES, by LEDOUX JR on "Electric activity of the labyrinth nerves of the frog", an excellent continuing of the classic experiments of LOWENSTEIN, SAND and HALLPIKE, by HAMBERGER on "Cytochemical investigations on N vestibularis", the beginning of biochemistry in otology MYGIND gave a lecture on "the static function of the labyrinth" sparing himself no pains by sending a beautifully illustrated report to all the members before the meeting

After the meeting many members went to Stockholm on invitation by HOLMGREN for a conference on audiology, the first in Europe The result from these Collegium activities was that in the following year, before the international congress in London, the first international congress on audiology was held. In this congress we heard much about the new activities in England There is no doubt that these early initials accomplished by the Collegium have been of paramount importance for the future development of audiology

Copenhagen is an extremely beautiful and gay city, but also science has always flourished there. It has sometimes been called the Paris and also the Athens of the North Next time in Rome in the holy year 1950 under the presidency of FERRERI we were of course very much impressed by the beauties of the eternal city, but only 51 members enjoyed it (politics!) There were 23 papers, 15 on the ear An enormous amount of work has been done by CAMBRELIN and collaborators, authorities in this field as ARSLAN, CRABBÉ, VAN EGMOND, HALLPIKE, PORTMANN on a report concerning "standardization and simplification of the examination of the vestibular apparatus" But it became evident that the time was not yet ripe for a standardization and medical men are very independent people, who are fond of their own methods! 10 years later we were again in Italy in Padua where under the presidency of ARSLAN we had an excellent symposium on the sensulae of the vestibular part of the labyrinth with DOHLMAN as a moderator

We mentioned the hospitality of our Scandinavian colleagues But with Scandinavians it is difficult to organize something during the summer months, when the "white nights" turn the mind to celebrating and holidaying In view of this it is all the more to be appreciated that MEURMAN enabled us to share this by organizing the Helsinki meeting early in July 1951 Many members travelled via Sweden, where we were cordially received by SKOOG in Stockholm and NYLEN in Uppsala The further trip to Finland was by way of Turku, SIIRALA made us welcome here, and a few of our members experienced the fascinating and to them new attractions of a sauna In Helsinki it was considered a suitable time for the Germans to rejoin us There were only 35 members but that was probably the cause of a very intimate atmosphere Another reason was the very kind reception by president and Mrs MEURMAN and our Finnish colleagues There were 21 papers with 37 discussions, an excellent paper on invitation was from our guests SAXEN and OJALA on "The genesis of attic cholesteatoma" We have often enjoyed guest lectures on invitation, mostly on work done in a laboratory in the place of the meeting

### The Presidential Address

In discussing the last Groningen meeting we mentioned that the first presidential address after the war was to be delivered by FERRERI in Rome (1950) With PIETRANTONI and ARSLAN from Padua, FERRERI was among the most prominent postwar Italian members He was of great stature, both physically and as an otologist, and his speeches were famous; he brought much gaiety into the Collegium At that memorable meeting in Rome where we lost such inferiority feelings — as we may have had — by having dinner in the guise of Roman senators, complete down to toga and laurel leaves, he told us about the Ospedale di Santo Spirito — the oldest hospital in the world (dating back to 1200)



Fig 9 Ward of The Ospedale di Santo Spirito, Rome
Votive altar and organ

In fact we held our meetings in this hospital, and lunched there every day. The next year in Helsinki, MEURMAN received us in a hypermodern clinic which had just been finished, and the difference makes us think of the past and the present. The hospital in the old Europe was built from charity, as an extension of the church; this is clearly demonstrated in fig. 9, showing the interior in one of the wards of the Ospedale di Santo Spirito, dating back to the 17th century and still in use. The patient's spiritual health was more important than the health of his body. In any case, what could a physician in the early years of the 13th century do for his patient? Today, with the example of American hospitals in mind, we are primarily concerned with efficiency. To phrase it differently: while formerly a hospital was designed for the patients, it is now designed for the doctor. And because the latter can do so much more than he used to, this is also for the benefit of the patient. But we cannot escape some discomfort thinking of a future with an ever more mechanized brand of medicine, with ever more computers doing much of the work now done by physicians and nurses. Now that personal contacts are becoming less and less important, it cannot do any harm to contemplate the charity of years gone by.

FERRERI's address was much appreciated; with it, he created the tradition of offering something of local colour. And this was bound to be fascinating, for the Collegium often met in places with a very rich history. GUŠIĆ in Zagreb discussed Yugoslavia — a country of transition between West and East and one greatly influenced by such diverse civilizations as the Roman, Byzanthine and Venetian. WILSON spoke about the "Dublin School of Medicine" with its many famous names, among which that of otologist SIR WILLIAM WILDE interested us above all. Nearly inexhaustible was the subject matter of HOFER's address on Austria's contribution to the evolution of O.R.L. during the period 1850-1910. ARSLAN discussed the discoveries in O.R.L. made during the 16th century in Padua; nearly all the great anatomists of the Renaissance have made their contributions in this respect. CHRYSSIKOS delivered an absorbing address on O.R.L. diseases in ancient Greek medicine. And in Würzburg we were impressed by a demonstration of the protocol of the first meeting in which ROENTGEN reported his discovery, whereupon president KÖLLIKER suggested that these rays be named Roentgen rays, as they still are today.

## ZURICH SEPTEMBER 1952

This important meeting under the presidency of RUEDI, was an ordinary meeting again, but one of extraordinarily great scientific value. There were 32 papers, and two communications on invitation which were of exceptional quality. Our guest VON MURALT reviewed "Modern physiological aspects of hearing", and HILDING from Duluth discussed "Some therapeutic considerations in the development of postoperative atelectasis of the lung", explaining the mechanism by which the air disappears behind the obstruction and emphasizing the importance of ciliary action. HILDING and a few others such as NEGUS and MESSERKLINGER saw to it that the deeper air passages were not too much neglected in the

Collegium HILDING did experimental work on many aspects of O R L His second paper on invitation (Bordeaux 1956) concerned "Cigarette smoke and carcinoma" and was again based on a splendid experimental study NEGUS was the only other member ever to read two papers on invitation, in 1954 (London) he read the second, on "The function of the paranasal sinuses", as expected, this led to a discussion with PROETZ — also an authority on the physiology of the nose

The problem of laryngeal paralysis was discussed once more by HOFER, referring to his wellknown studies of retrograde degeneration, CLERF treated the same

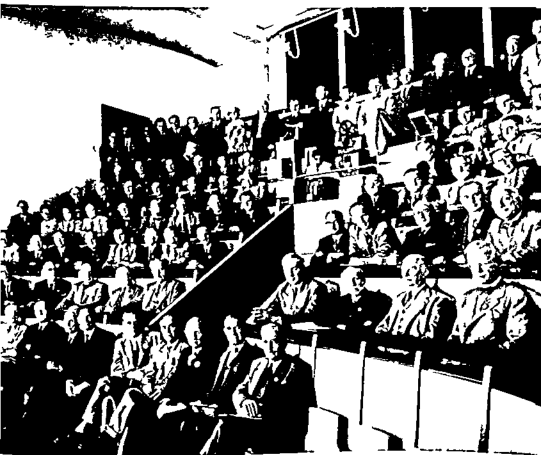


Fig 10 Zurich September 1952
Prosperous Collegium 94 honorary members and members present

subject on the basis of an unusually extensive experience BATEMAN presented a fine analysis of post laryngectomy oesophageal speech

There were 94 participants — a record attendance not likely soon to be surpassed No fewer than 7 American members attended, 6 gave a paper of good scientific standard and on very different subjects, which is appreciated in the Collegium The names of CLERF and HILDING have already been mentioned, HOLINGER discussed the congenital anomalies of the bronchi ', LINDSAY gave an outstand

ing paper on experiments on obliteration of the ductus endolymphaticus in the cat, GUILD discussed the hearing in partial section of the cochlear nerve operated by the famous neuro-surgeon DANDY, FOWLER did experiments on cats and rabbits studying "neurovascular hypersensivity to symptoms and diseases" For the first time after the war 10 German members were amongst us, mostly new ones, but also some old members and such good friends as VON EICKEN, who like ECKERT MÖBIUS did wonderful work for the Collegium friends in occupied countries during the war

The atmosphere was very gay and friendly from the very start, when on the first evening we were the guests of MR and MRS RUEDI in their fine house on Dolderberg A peak of merriment was reached when a wellknown Harley Street specialist could no longer resist the attraction of a small pond and, earnest and dignified as only an Englishman can be, took to the water

The Collegium had thus completely resumed its activities, and its further life need only be briefly discussed In 1954 we went to London, where NEGUS doubled in brass as president and treasurer There were 68 members and, to demonstrate how faithfully the meetings in the Collegium were attended, we mention that 61 members were present both at the opening and at the closing session The next year brought news in that we met at two places, Zagreb under the presidency of GUŠIC and Beograd under PODVINEC It has been pointed out that the so called small countries have always shown a great interest in the Collegium This was emphasized again in Zagreb of the 44 members, 6 came from Sweden 5 from Belgium, Netherlands and Switzerland each The 1956 Bordeaux meeting has already been discussed In 1958, WILSON presided in Dublin, in 1959 HOFER in Vienna, in 1960 ARSLAN in Padua, in 1962 CHRYSSIKOS in Athens, in 1963 SIMSON HALL in Edinburgh and in 1964 WULLSTEIN in Wurzburg It were almost always meetings with a high scientific standard, with very pleasant receptions and sight seeing and much appreciated intimate at homes as in the houses of WILSON and ARSLAN

All meetings have now been reviewed Those held after the war have not been discussed in detail because they have not yet entered the realm of history Many members have attended them, and it is sufficient merely to mention these occasions to revive the memories of the many events which enlivened these meetings This does not apply to the pre-war meetings, the majority of the then time members have passed away And the few who are still alive have reached an age at which one no longer blushes Of course it is impossible to give a really comprehensive account of these earlier meetings and it would be boring to discuss all papers read We have merely mentioned a few to give the younger members an impression of past events in the Collegium Surely they must have noticed that a great tradition has been maintained, and that little has changed in the organization and essential features of our meetings Only new names crop up Life goes on and the older members see with pleasure that so much good has been handed down to a younger generation

We may mention that RUEDI in 1950 succeeded NEGUS as treasurer, while gener

al secretary HUIZINGA was succeeded by JONGKEES in 1960 And we must also mention, with deep regret, some of the really faithful who have left us since the war DE KLEYN (1949), VON GYERGYAY and LEIRI (1952), HOLMGREN (1954), BALDENWECK and BURGER (1957), VAN GILSE, NAGER and VOSS (1959), MEURMAN, VON EICKEN and WILKINSON (1960), FERRERI, HICGUET and SOURDILLE (1961), in 1962 we lost BOUCHET, an exceptional mind and a good friend, in 1964, much too young, FOWLER departed, he was the son of a great otologist and himself a man of many original ideas

HEREDITY IN O R L AND THE SHAMBAUGH PRIZE

The passage of 40 years means the arrival of a new generation, as clearly indicated in our membership list Oto Rhino Laryngology is an attractive discipline, as demonstrated by the remarkable fact that no fewer than 8 of our members were joined by their sons as Collegium members, and the younger generation, too, includes some prominent men of our profession There are the PORTMANNS, father and son, from Bordeaux, at the Zagreb meeting in 1955, even before he was a member, MICHEL PORTMANN was invited to read a paper on "The terminal fibres of the auditory nerve" There are the OPPIKOFERS from Basel, the MEURMANS from Helsinki and Turku, respectively, the LEEGAARDS from Oslo, the DEMETRIADES from Athens (first members from Greece), the BRUNETTIS from Venice and Turin, respectively, the VON GYERGYAYS from Cluj, the father was our first Rumanian member and a great authority on the pathology of the nasopharynx, on which he read a number of papers Last but not least there are the SHAMBAUGHS from Chicago

GEORGE E SHAMBAUGH SR has been mentioned as one of our first members from the USA, and as the first American member to read a paper on invitation (London 1929) He was *trained in* O R L in Berlin and some of his early scientific papers were written in German, as a young man, he already showed an international orientation He often spoke of his very fond Collegium memories, and it was "because of his love for this organization" that, after his death in 1948, his six children, probably on the initiative of GEORGE E SHAMBAUGH JR (mentioned *in connection with the extraordinary meeting* in Brussels), decided to establish the "Shambaugh Memorial Trust Fund', which every other year provides the SHAMBAUGH PRIZE for outstanding work on the ear The first prize was awarded in 1949 to VON BEKESY Subsequent prize winners were CAUSSE (posthumously), HALLOWELL DAVIS HALLPIKE, WEVER, LINDSAY, RÜEDI, HILDING and DOHLMAN *The candidates were suggested by* the Collegium and anyone with an understanding of O R L who reads the list will recognize that the choices have been very felicitous

# OTO-RHINO LARYNGOLOGY AND THE COLLEGIUM AFTER WORLD WAR II

After the last war, O R L underwent perhaps even greater changes than other specialties In the past, otitis media was the most frequent cause of death in our clinics, but since the introduction of antibiotics this inflammation is no longer fatal It has been supposed that activities would diminish as a result, but the opposite has occurred operations improving hearing were developed Otosclerosis has been discussed, our members WULLSTEIN and ZOLLNER have done pioneer work in the field of tympanoplasty Operative technique was revolutionized with the introduction of microsurgery (NYLÉN introduced this new art in our special field), and it has become the ear surgery par excellence for the younger generation The splendid and new feature of this surgery was that it considered function and offered what might be called physiological operations — a trend also observed in other fields

The chisel was the principal instrument of the otologist of past generations It has even been stated that "the chisel is the extension of the finger, the sculpturer creates art with a chisel, not with a drill, the dentist drills but the otologist chisels" But there is nog longer any truth in this statement Some of the older colleagues found it difficult to adjust themselves and may even have become somewhat tragic Striking is what CHEVALIER JACKSON once said "It is hard to teach old dogs new tricks" But unheard of possibilities opened up for the younger generation

Much of major otological surgery (e g in sinus thrombosis and other complications) has become obsolete, on the other hand, major operations summarized under the heading of head neck surgery became more and more the domain of O R L In addition there was considerable expansion due to the developments in audiology, phoniatrics, allergy, otoneurology, broncho oesophagology and plastic surgery, which has become clearly manifest in the enormous increase in the number of patients in O R L clinics Now one may object that "patients are nothing to do with the Collegium", but such an objection overlooks the fact that science in medicine *is not a sterile science It is so absorbing precisely because, in the background,* there is always the thought "it might benefit the patient" This was probably what PASTEUR meant by his famous statement "La science se fait non seulement avec l'esprit mais aussi avec le coeur" ("Science is made not only with the mind but also with the heart') Now how did these major changes affect the Collegium?

It has been pointed out that the scientific interest primarily focuses on the ear, i e the organs of hearing and equilibrium with their correlates in the CNS There were 10 ordinary meetings before and 10 after the war, and this facilitates a comparison The total numbers of papers read before the war was 221, of which 148 dealt with the ear (i e two-thirds), and 73 discussed other subjects After the war there were 306 papers read, including 205 about the ear (again two-thirds) and 101 about other subjects The distribution has remained strikingly constant Of course it is not surprising that in spite of the expansion of the medical field, the interest

in the ear has not diminished This must be ascribed in the first place to audiology and copho surgery The latter revived the interest in the mechanism of hearing and opened up new avenues of investigation, e g the study of the perilymph in surgical patients, which at the last meeting was the subject of papers by RÜEDI and WULLSTEIN with their co workers New achievements such as the use of radioactive isotopes were applied PORTMANN and co workers discussed these as early as 1951 (Helsinki), and DOHLMAN and ORMEROD did excellent work in this field (Dublin 1958) Experimental work on ultra sound was discussed in Padua (1960) by ANGELL JAMES, ARSLAN, FORMBY and SJÖBERG ENGSTROM discussed the use of the electron microscope as early as 1952 in Zurich and read a paper on invitation on this subject in London (1954)

The new apparatus called for cooperation with histologists and supplied considerable new information on the ultra structure of the sensory spots in the labyrinth This was of importance to physiology — the subject of numerous animal experiments New points of view arose especially from the ever increasing applications of electronic techniques Even before the war (DAVIS and SAUL) it had been established that the potentials demonstrated according to WEVER and BRAY had two components the action potentials from the VIIIth nerve, and the microphonics It was found that these could also be derived from the sacculus (ASHCROFT and HALLPIKE), after the war this was also found possible, under special conditions (TULLIO), from the crista (BLEEKER and DE VRIES) Papers on this subject were read by VAN EYCK and ARDOUIN, among others

VAN EYCK later also made use of the electromyogram, which was also applied in laryngeal physiology Another important finding dating back to pre war days was the spontaneous activity of the vestibular sensory spots (LOWENSTEIN and SAND), variable by stimulation This was the subject of a paper by LEDOUX, on the basis of personal experiments Another promising field is the chemical investigation of endolymph (our member VILSTRUP) Thus we continue to hope that the mechanism by which mechanical energy is converted into electrical energy in the ear will one day be clarified Perhaps this hope is too closely linked with life, which we do not yet understand But as GOETHE already put it "Der Mensch muss bei dem Glauben verharren, dass das Unbegreifliche begreiflich sei, er wurde sonst nicht forschen" ("Man must persist in believing that the incomprehensible can be comprehended, otherwise he would never investigate")

The electron microscope was also used in more precise studies of the nasal mucosa (BURIAN, Athens) It can be observed that in recent years there is a growing interest in the important pathology of the mucosa of the upper and lower air passages This is demonstrated in animal experiments with microscopy and in human studies such as those of MESSERKLINGER, MAJER HLAVACEK, BURIAN, GUŠIĆ and co-workers In addition to histology, chemistry also yielded important information in this respect

Audiology developed as one of the few positive direct consequences of war The invention of midget type valves made it possible to design our modern small size I
which the

already a film on this subject There was the rapid rise of electro acoustics, on which many papers have been read in the Collegium

Electro-techniques were more and more important to man, not only for the acoustic but also for the vestibular part of the labyrinth There was nystagmo graphy, first discussed by MITTERMAIER (a great authority on labyrinthine physio logy in Germany, like our member FRENZEL) in London in 1954, and subsequent ly often reported on by MONTANDON, JONGKEES, LACHMANN and GREINER Elec tro techniques were also applied in laryngology and phonetics, as shown in papers read by TATO in London (1954), PORTMANN in Zagreb (1955), GREINER in Vienna (1959) and KRMPOTIC in Athens (1962) All this required an increasing array of apparatus and called for more and more assistance from experts

From the start, the Collegium has been fortunate in having the support of theorists Members past and present include the physiologists ZWAARDEMAKER, TAIT, TULLIO, DI GIORGIO, HALLOWELL DAVIS, KEIDEL, DESMEDT, the anatom ists LORENTE DE NO, GUILD, DE BURLET, KRMPOTIC, the physicists VON BEKESY, HUIZING and GROEN There are two women among these members, DI GIORGIO from Turin was a great authority on the CNS, who read a paper on invitation on "Relations between vestibular activity and the cerebral cortex" (Rome 1950) KRMPOTIĆ from Zagreb has done work on the larynx and ear which has attracted great attention in recent years A third woman — DIDA DEDERDING — was a faithful assistant to MYGIND for many years ZANGE's (Jena) pupil and successor ALBRECHT was accepted as a member in Tokyo DEDERDING died in 1955 and DI GIORGIO in 1961, and the anatomist KRMPOTIC and otologist ALBRECHT are therefore at present the only ladies who adorn our ranks

The help given us by theorists is likely to increase in future, it is the consequence of the rapid evolution of technologies with ever more complex apparatus, and the progress made in physics and chemistry In fact the ultimate solution of many problems (e g the function of hearing and of the organ of equilibrium) lies in the field of either physics or chemistry Science is becoming so intricate that individual one man research is becoming more and more impracticable, teamwork is increas ing, as this has its reflections in the papers read in the Collegium Of the 221 pre war papers 200 were given by a single colleague Of the 306 postwar papers, only 170 were single-author papers while no fewer than 136 had two or several authors, often including a theorist These figures of course indicate a minimum, many more papers must in actual fact have been prepared with the help of others

War has had one other unmistakable result Of the 221 pre war papers, 53 (24%) were read in English, 61 (28%) in French and 107 (48%) in German Of the 306 postwar papers 160 (52%) were in English, 79 (26%) in French and 67 (22%) in German

## THE PRESENT AND THE FUTURE

The Collegium was founded with the object of discussing scientific problems of O R L at regular meetings in a spirit of friendship Has the Collegium fulfilled this goal? Any older member will wholeheartedly confirm this question But this account of the fortunes, trials and tribulations of the Collegium was not written merely to revive pleasant memories in the older members, it was written chiefly for the younger colleagues, in the hope that they, too, will be convinced of the great tradition of the Collegium

It was written to give an indication of the high scientific level of the communications, which have very often discussed new and important findings It was written also to elucidate the significance of the "Amicitiae Sacrum"

The last meeting in Wurzburg (1964) was especially successful in both respects, 84 members from 22 countries attended All must have gone home with a sense of satisfaction President WULLSTEIN's cordial hospitality started on the evening before the meeting with an at home in his splendid house There were numerous papers at a very high scientific level Not all of them can of course be mentioned, but an exception must certainly be made for the 3 papers on invitation The physiologists DAVIS and KEIDEL discussed slow cortical responses evoked by acoustic stimuli — a new subject in neuro otology and electrophysiology on which KEIDEL had already reported in Athens Our guest from Boston, NELSON YUAN-SHENG KIANG, presented an impressive paper on "Stimulus coding in the auditory nerve and cochlear nucleus" On the following morning we saw very succesful demonstrations in KEIDEL's laboratory in Erlangen All this once again emphasized the importance of the fact that the Collegium has always maintained such good relations with theorists

The excursion to Erlangen was followed in the afternoon by a visit to castle Weissenstein with the ladies The two events were scheduled on a separate day, bringing the Wurzburg meeting to a total of 4 days This was necessary, for there were 37 papers, divided over 3 days This called for two afternoon sessions, contradicting the agreement made earlier in Frankfurt (1930), to keep the afternoons free for excursions

At the foundation and later again it has been emphasized that the meetings of the Collegium were to differ from other meetings in that communications and discussions were to be given ample time The founders already wrote "the number of papers must be limited" The number of papers has varied rather widely In Edinburgh there were 24, in Athens no fewer than 41, in Padua 31 and a symposium, in Vienna 38 and in Dublin 24, there were never too few papers The Collegium is not likely in the near future to fade away because of a deficiency of papers contributed, in fact an overcrowded programme seems a graver risk The Committee must even more strictly select papers, and may have to disappoint some members, but this is necessary if the high scientific standard of the Collegium is to be maintained This has already been discussed in a business meeting, and proved to be a point of complete agreement

Otherwise, as we muse about the future of the Collegium, there is nothing

against which one might warn, and nothing that one would see changed One can only say "continue on this road" The Collegium is a flourishing organization which is fortunate in that its management is in excellent hands

According to the latest list (1963) there were 228 honorary members and members, from 41 countries Of these countries, 22 are in Europe, 11 in the Americas, 4 in Asia and 2 in Africa and Australia The last country to be admitted was Japan Member FUKUDA delivered an outstanding paper on vestibular training in fowls (Athens), other Japanese members are GOTO, KIRIKAE and ONO, the general secretary of the Tokyo VIIIth International Congress, 1965 As usual a business meeting of the Collegium was held during this congress, as it had been done in London (1949), Amsterdam (1953), Washington (1957) and Paris (1961), in more recent years with a lively luncheon to follow

Speaking about business meetings, the influence of the Collegium is clearly manifested in the fact that our friends across the Atlantic have in the past few years organized a business meeting during every Pan American Congress, the chief purpose has been a friendly discussion, which always culminated in much appreciated greetings to the general secretary Much friendship indeed has been established on international lines in the small world of the Collegium The world's hopes for the future might be brighter if there were a Collegium Diplomaticum Politicum Amicitiae Sacrum!

In 1966 we shall travel for the third time to France, to a Lyons meeting presided by MOUNIER KUHN — a many sided personality in O R L Many members look forward to this meeting with pleasure, and it is bound to be another good meeting indeed There need be not a shade of fear for the future of the Collegium Oto Rhino Laryngologicum Amicitiae Sacrum

Groningen, 1966

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# DISCRIMINATION PERFORMANCE OF HIGH SCHOOL SOPHOMORES ON A BATTERY OF AUDITORY TESTS

BY

DONALD N. ELLIOTT,
WINIFRED D. RIACH, JOHN P. SHEPOSH,
and CONSTANTINE TRAHIOTIS

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*Auditory Research Laboratory, Wayne State University,*
*Detroit, Michigan, U.S.A.*

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 216

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# ABSTRACT

One of the most pressing needs in the area of auditory rese
establishment of stable representative data on various types (
discrimination Consequently 400 normal hearing subjects (Ss
ministered a battery of discrimination tests The battery consi
pure tone tests and one speech discrimination test The par
the pure tone stimulus investigated were frequency (monotic an
intensity (monotic and dichotic) and duration (monotic only)
crimination data together with background data of the sample
mitted to multiple regression and factor analyses In considerin
ings three aspects of the data are of particular interest First, t
level of performance with the exception of the duration tes
nificantly poorer than data usually reported Secondly the
reliability varied greatly from test to test even though the
presenting the stimuli remained unchanged Finally while a s
discrimination factor appears to exist it is clear that vari us
tion tests tend to be relatively independent of each other

# INTRODUCTION

In the investigation of auditory discrimination, most studies have been parametric in nature, concerned primarily with the manner in which discrimination changes as various aspects of the signal are altered, or as psychophysical test procedures are varied The studies of Shower and Biddulph (1931) and Riesz (1928), for example, investigated in detail the effects of signal frequency and signal intensity upon discrimination These studies were directed primarily toward the establishment of general laws describing such psychophysical relationships, and as such are landmarks since the data reported have been found to hold generally for most listeners

In developing these general relationships, it has been the custom to use only a few well-trained Ss and to test them under all the possible combinations of the parameters being investigated Examination of these studies, then, yields little information on the performance to be expected from a nonpracticed normal hearing S since the influences of motivation, experience, and other nonauditory factors are held to a minimum Further, the range of discrimination ability which can be considered normal is likewise in question It is with these aspects of discrimination ability that the present study is primarily concerned

It should be noted that very few studies have been concerned with the determination of normative auditory discrimination data, consequently, studies such as those mentioned above are usually cited in any discussion of the ear's ability to discriminate frequency or intensity differences often with the implication that they represent, in fact, 'normal' discrimination Anyone, however, who has had experience in auditory testing soon becomes painfully aware of the tremendous variations in performance among the Ss who are audiometrically normal particularly in the determination of differential thresholds Consequently, the use of averaged data based on small groups of well-practiced Ss is of little practical use in defining normal discrimination, rather, normal ranges of discrimination based on large randomly selected samples of Ss are needed

The plan of the present study has been to obtain a relatively large representative group of Ss with normal audiograms and with no history of traumatic auditory experience or damaging auditory diseases, and to test each of these Ss on a battery of discrimination tests From these data, ranges of performance on each of the tests have been determined as a means of establishing normal discrimination It was expected of course, that performance would on the average, not be as good as that reported heretofore in many psychophysical studies since it involves the testing of

unpracticed Ss whose motivation in some cases could not be expected to match that of selected Ss utilized in many psychophysical studies

In addition to the determination of auditory discrimination norms, this study has also concerned itself with the relationships between performance on the various discrimination tests and, in addition, the relationships between certain nonauditory factors and performance on the discrimination battery We expected to utilize the relationships as indices of the extent to which (1) common auditory discrimination factors exist, and (2) the extent to which nonauditory factors are reflected in auditory performance

# THE SAMPLE

Since normative discrimination data were to be obtained it was necessary that a large number of Ss having normal audiograms and no history of injuries to the ear or hearing difficulties be tested Additionally it was clear that a heterogeneous sample in which selective factors would be minimally reflected should be utilized Further it was felt that the sample should reflect physiologically mature auditory functioning Consequently it was decided to use high school rather than university students since the latter group is obviously a more highly selected group Even high school students in their third and fourth years represent a selected group since dropping out of school is permitted at 16 years of age On the basis of these considerations it was decided to use high school sophomores as Ss since as a group they would mirror the adult population more closely than a younger group and still be highly heterogeneous While such a sample reservoir was not ideal in the sense that it was completely unaffected by selective factors it appeared to approach this goal reasonably well

Arrangements to carry out the selection of Ss were made with the Detroit Board of Education and the Catholic Archdiocese of Detroit With their aid and cooperation the names of all sophomores in the public and parochial schools were made available There were approximately 19 000 such students 13 950 sophomores from 22 public high schools and 5615 sophomores from 51 parochial schools An initial sample of about 4% of this group was drawn at random this gave us more than the 400 Ss we expected to test eventually It was necessary to draw the large initial sample since some would be unwilling to cooperate or would be excluded because of heaing losses

After the initial random selection a letter was sent to each student s parents describing the study and requesting permission to test the student Accompanying this request was a questionnaire which served as a screening device to determine whether there was a history of any injury or disease that would impair the student s hearing The parents were informed that the Ss would be paid a nominal fee plus bus fare for participating Parents who refused or did not respond were if possibl contacted by telephone Permission to test the student was received from approximately 70% of the parents contacted Of the remaining 30% outright refusal occurred in one half of the cases while student dropouts transfers and nonresponders accounted for the remaining cases Among the students eventually called in for testing about 4% could not be used because of hearing losses and

unpracticed Ss whose motivation in some cases could not be expected to match that of selected Ss utilized in many psychophysical studies

In addition to the determination of auditory discrimination norms, this study has also concerned itself with the relationships between performance on the various discrimination tests and, in addition, the relationships between certain nonauditory factors and performance on the discrimination battery We expected to utilize the relationships as indices of the extent to which (1) common auditory discrimination factors exist, and (2) the extent to which nonauditory factors are reflected in auditory performance

TABLE 3. *Occupational level of parents*

| | Percentage of sample |
|---|---|
| Unemployed | 5.25 |
| Unskilled laborers | 22.0 |
| Semi skilled | 29.75 |
| Skilled | 14.5 |
| Supervisory, minor sales, minor proprietory | 15.5 |
| Higher supervisory, major sales | 5.25 |
| Managerial | 2.75 |
| Professional major proprietory | 5.0 |

TABLE 4 *Musical training*

| Number of years | Percentage |
|---|---|
| 0 | 62 |
| 1 2 | 18 |
| 3–4 | 10 |
| 5–6 | 7 |
| More than 6 | 3 |

compared, no significant differences were evident Although such evidence concerning the similarity of the students in the two groups is tenuous, it does not appear that any very great differences existed between the two groups of students

In order to obtain information on the general nature of our sample of Ss, several measures were obtained These included scholastic aptitude, experiential background, and familial background Data on scholastic aptitude was available from school records, data on the other items was obtained from a short interview with the S at the time of testing Data on these various factors appear in Tables 1 through 6

TABLE 5 *Amount of time foreign language spoken in Ss' homes (Based on Ss' judgment)*

| Amount of time | Percentage of sample |
|---|---|
| No other language | 81.00 |
| Less than 25% | 9.5 |
| 25 50% | 2.75 |
| 50 75% | 2.00 |
| 75 100% | 1.75 |

TABLE 1 *Comparison of sample and national norms for SRA[a], STEP[b], SCAT V and Q[c].*

| Sample mean | | National norms (mean) |
|---|---|---|
| *Catholic school students* | | |
| SRA | 110 | 106 8 |
| *Public school students* | | |
| STEP | 287 | 289 |
| SCAT V | 276 | 279 |
| SCAT Q | 291 | 293 |

[a] Science Research Associates Inc High School Placement Test National norms refer to HSPT variable, educational ability (form 63k)

[b] Educational Testing Service Sequential Tests of Educational Progress—Reading Subtest Norms obtained from 1963 supplement

[c] Educational Testing Service School and College Ability Tests—Verbal and Quantitative Tests Norms obtained from 1963 supplement

about 1% could not be tested because of inability or unwillingness to follow test instructions The final sample was then chosen at random from those replies in which testing permission was given and which indicated normal hearing

Since the selection procedure was designed to obtain a sample of Ss which would be representative of young persons with normal hearing, we were concerned with the possible biasing effect of our failure to obtain permission to test 30% of the sample as originally drawn Only two types of information were available for comparing the students who did come in for testing and those who did not These were the scholastic aptitude scores which were available for all students and the socioeconomic level of the neighborhood in which the students resided When these were

TABLE 2 *Total years of education of Ss' parents*

| Number of years | Percentage of sample |
|---|---|
| 10 and less | 2 |
| 11 15 | 13 |
| 16 20 | 24 |
| 21 25 | 43 |
| 26–30 | 12 |
| 31 35 | 5 |
| 36–40 | 1 |

*Note* —Total number of Ss whose parents both completed high school 139 (35%)
Total number of Ss whose parents both completed college 17 (4%)

# PROCEDURE

## AUDITORY TESTS

*Pure tone discrimination test procedure*

Several considerations determined the procedure to be followed in obtaining pure tone discrimination thresholds First since 400 students were to be tested on a battery of discrimination tasks it was not feasible to test each one individually Second the Ss were not practiced in auditory discrimination tasks and were available for only one session Third it was expected that the students would vary substantially in their ability to understand any testing procedure and/or in their willingness to try to understand and follow it Fourth it has been observed that listeners differ greatly in their willingness to report differences even when detected in observed signals near threshold level

On the basis of these considerations it was decided to utilize a two alternative forced choice procedure in which two successive trains of five tone pulses each comprised a trial Of these two pulse trains one contained only standard tones while the other contained tone pulses which alternated between standard and comparison (see Table 7 for details) Intertrial intervals were five seconds and there was a 30 second rest period after each block of 16 trials It will be noted from the diagrams in Table 7 that the first third and fifth tones of the train containing the comparison tones were standard pulses Thus all pulse trains started and ended with the standard tone

The S was required to report which of the two trains of pulses contained tones which were alternately different by recording his judgment on an answer sheet after each trial With this procedure Ss were not required to make judgments of higher or lower in the frequency discrimination tests or of louder or weaker in the intensity discrimination tests It was hoped that the use of the neutral judgment of different would avoid semantic difficulties which might result from the use of the other terms We found through preliminary testing that this procedure was easy to understand regardless of the nature of the discrimination and that the Ss were able to perform satisfactorily after only a few practice trials[1]

[1] It should be mentioned that before settling upon the specific characteristics of the trials indicated above several pilot studies were carried out In these studies the number of pulses, pulse length and interpulse interval were varied in an attempt to arrive at the optimum values (in terms of threshold size) While several such studies were carried out it should be emphasized that there was insufficient time to investigate

purpose a test of tonal duration was developed Further it appeared *desirable to include tests involving both ears For this purpose two dichotic* discrimination tests were developed these involved presentation of the comparison tones in only the right ear the comparison tones in one test varying in frequency and in the other test in intensity In summary the five pure tone discrimination tests were

Monotic frequency discrimination
Dichotic frequency discrimination
Monotic intensity discrimination
Dichotic intensity discrimination
Monotic duration discrimination

Diagrams of these tests are presented in Table 7

*Verbal discrimination test*

It was felt that in addition to the pure tone discrimination tests a test of verbal discrimination should be included since in many respects such discriminaton represents the ultimate task the auditory system is called upon to perform and is of great practical significance Here again the fact that Ss were to be tested in groups made it necessary to record individual responses for later scoring further since we were not interested in measuring spelling or writing ability it appeared desirable to make the S s responses as simple as possible It was therefore decided to utilize Fairbank s Rhyme Test in which the S is asked to print the first letter *of each word he hears the stem of the word already appearing on his* answer sheet The test used was duplicated from the original test prepared by Fairbank s with broad band noise added It consisted of five subtests of 50 words each with similar stems but different starting consonants Details of the test and its construction can be found elsewhere (Fairbank s 1958)

Since we were interested in determining not only the S s overall verbal discrimination but the slope of his articulation function each successive subtest was presented with increased noise levels (Table 25) the verbal material being maintained at a constant level of 40 dB above each S s threshold of audition (See Fairbank s for a description of the manner in which verbal level was specified )

TESTING PROCEDURE

*Thresholds of audition*

At the start of the session each S was tested to determine whether his thresholds of audition w normal at octave intervals from 125 through 8000 cps f both ears ex at 1000 cps no attempt was made to deter mine th threshold if better than audiometric zero At 1000 cps however reshol ations were for each ear since this thres used in ie intensity crimination signals

for this purpose, the audiometer was modified so that hearing levels of up to −30 dB could be measured at 1000 cps

The criterion for accepting a S for discrimination testing was (1) that his threshold at 1000 cycles not exceed audiometric zero by more than 2 dB, and (2) that his hearing loss at any other frequency not exceed 10 dB In all, 32 Ss were rejected because of hearing losses This represents about 7% of the total number of Ss reporting for the test

*Pure tone discrimination tests*

As indicated earlier, the length of the test battery and the size of the sample to be tested required that the testing be carried out with several Ss simultaneously Facilities for testing four Ss at once were therefore developed, with the Ss seated in the same test chamber. There were separate intensity controls for each earphone of each S so that all pure tone standard signals were presented 40 dB above each ear's threshold of audition

Each discrimination test was preceded by eight practice trials similar to the first block of trials, and all Ss' performances on these practice trials were checked before the test was continued If any errors were made on the practice trials, the trials were repeated and responses again checked, this was done a third time if errors were still evident After three presentations of the eight practice trials, however, the test was continued, even if errors might still be evident for a S

The order of test presentation was of some concern since it appeared quite possible that practice and/or fatigue effects might develop as the testing session progressed In view of this, it was decided to vary in a systematic manner the order of presentation of the five pure tone discrimination tests so that each test occurred equally often at each of the five possible test positions Thus with 400 Ss, e g, Test A was presented in test positions one through five 80 times each, further, for the eighty times that Test A occurred at any given test position, the remaining four tests were systematically arranged so that each was presented 20 times at each of the remaining test positions A further control in the ordering of the tests involved setting up the test orders so that each test preceded and followed every other test an equal number of times With this procedure, it was possible to determine whether position was a significant factor in mean performance on each test

A second concern was with the reliability of the discrimination test scores as well as possible specific improvement that might occur as a result of the testing Consequently, whenever a test occurred first in the testing sequence, it was immediately repeated Thus, since each of the five pure tone tests occurred in the first test position 80 times, test-retest reliabilities based on 80 Ss could be determined Further, as an indication of the extent to which performances improved as a result of practice, shifts in performance between test and retest could be evaluated

*Verbal discrimination test*

This test consisted of five subtests of 50 identical word stems printed on the Ss answer sheet. For each word stem, however, the starting consonant differed in each of the subtests.

The intensity level of the verbal material (mean VU meter readings) was presented 40 dB above the Ss 1000 cps threshold of audition for all subtests. The words were presented at a rate of one word each five seconds with a 30 second rest period between each subtest. Signal noise ratios decreased for each subtest as indicated in Table 2b.

Here again, there was a question of whether the other discrimination testing might generalize to verbal discrimination performance. While the possibility appeared slight, it was decided that the verbal discrimination test would be presented before the pure tone discrimination tests for half of the Ss and following these tests for the remaining Ss.

*Apparatus*

In developing the various discrimination tests, it was recognized that while it would be possible to develop a system of sound generating and control apparatus that could be rapidly altered for presentation of various pure tone discrimination tests, the problems of constantly checking out such systems with its many components and of rapidly detecting malfunctioning and trouble shooting it were indeed formidable. As an alternative which appeared worth considering, the recording of the discrimination tests on tape was investigated. It became clear immediately that direct recording AM recorders generally available were not stable enough, particularly with respect to signal intensity, and so FM recorders were considered. While such recorders are limited as to their upper frequency response and in some models have a (relatively) low signal/noise ratio, such limitations did not appear to be overpowering in the present situation in which the pure tone signals did not exceed 1000 cps in frequency and where narrow band pass filters could be introduced between the tape recorder and the Ss earphones.

On the basis of preliminary testing, it was decided to use a Honeywell 8100 FM recorder. Details of the functioning of this tape recorder appear in Appendix A. It is sufficient here to indicate that variations in intensity of output were not measurable, that random phase shifts approximated about one part in 20,000, and that the overall level of the distortion products was less than 2%. On the basis of these measurements, we believe that the results obtained with the taped discrimination tests would not differ from those obtainable with "live" equipment.[3]

[3] In this connection, a preliminary study was conducted in which intensity discrimination thresholds were obtained on 21 sophomore Ss using both live and taped signals. Results were: mean IDT for live presentation = 3.54 dB; mean IDT for taped presentation = 3.48 dB.

# FREQUENCY DISCRIMINATION

Both monotic and dichotic frequency discrimination thresholds were determined. However, since they undoubtedly did not provide the same cues for discrimination, the Ss' performances on the two tests will be considered separately.

## Monotic Frequency Discrimination (MFD)

The general test procedure for all pure tone discrimination tests is described above. From Table 7 it will be noted that the monotic frequency discrimination (MFD) test consisted of six blocks of trials, all tones presented to the right ear. The difference in frequency between the comparison and standard tones was reduced after each block of 16 trials. As indicated, the frequency differences ranged from 15 to 1 cps (i.e., the comparison tones ranged from 1015 to 1001 cps). This range was expected to encompass nearly all of the Ss' thresholds, defined as the frequency differences correctly detected on 75% of the trials of a block of 16.

### Results

It will be recalled that with five pure tone discrimination tests each test was presented in positions one through five an equal number of times. Since there was a question of whether effects of practice on preceding tests or of fatigue, boredom, etc., might affect performance on the MFD test

TABLE 8. *Mean test scores and standard deviations for monotic frequency discrimination test for each test position. Each mean is based on N = 80*

| Test position | Mean (cps) | Standard deviation (cps) |
|---|---|---|
| 1 | 11.46 | 8. 6 |
| 2 | 11. 2[a] | 21.95[a] |
| 3 | 9.48 | 5.80 |
| 4 | 8. 8 | 5.18 |
| 5 | 9.17 | 5.37 |

[a] One S was unable to differentiate frequency differences up to 200. Therefore her score was arbitrarily set at 200 (see Fig. 1). If this S is eliminated, the results for test position 2 are: Mean = 9.22, S.D. = 5.33.

*Verbal discrimination test*

This test consisted of five subtests of 50 identical word stems printed on the Ss answer sheet For each word stem however the starting consonant differed in each of the subtests

The intensity level of the verbal material (mean VU meter readings) was presented 40 dB above the Ss 1000 cps threshold of audition for all subtests The words were presented at a rate of one word each five seconds with a 30 second rest period between each subtest Signal noise ratios decreased for each subtest as indicated in Table 2b

Here again there was a question of whether the other discrimination testing might generalize to verbal discrimination performance While the possibility appeared slight it was decided that the verbal discrimination test would be presented before the pure tone discrimination tests for half of the Ss and following these tests for the remaining Ss

*Apparatus*

In developing the various discrimination tests it was recognized that while it would be possible to develop a system of sound generating and control apparatus that could be rapidly altered for presentation of various pure tone discrimination tests the problems of constantly checking out such systems with its many components and of rapidly detecting malfunctioning and trouble shooting it were indeed formidable As an alternative which appeared worth considering the recording of the discrimination tests on tape was investigated It became clear immediately that direct recording AM recorders generally available were not stable enough particularly with respect to signal intensity and so FM recorders were considered While such recorders are limited as to their upper frequency response and in some models have a (relatively) low signal/noise ratio such limitations did not appear to be overpowering in the present situation in which the pure tone signals did not exceed 1000 cps in frequency and where narrow band pass filters could be introduced between the tape recorder and the Ss earphones

On the basis of preliminary testing it was decided to use a Honeywell 8100 FM recorder Details of the functioning of this tape recorder appear in Appendix A It is sufficient here to indicate that variations in intensity of output were not measurable, that random phase shifts approximated about one part in 20 000 and that the overall level of the distortion products was less than 2% On the basis of these measurements we believe that the results obtained with the taped discrimination tests would not differ from those obtainable with live equipment [3]

[3] In this connection a preliminary study was conducted in which intensity discrimination thresholds were obtained on 21 sophomore Ss using both live" and taped signals Results were mean IDT for live presentation = 3 54 dB mean IDT for taped presentation = 3 48 dB

TABLE 10 *Summary of performance on the monotic frequency discrimination initial test, retest, and all test positions combined*

| | 10th percentile (cps) | 50th percentile (cps) | 90th percentile (cps) | Diff (90th 10th) (cps) |
|---|---|---|---|---|
| Test presented in first position of the battery sequence N = 80 | 3.5 | 8.5 | 25.1 | 21.6 |
| Retest immediately following first presentation N 80 | 3.0 | 8.7 | 20.3 | 17.3 |
| $r_{test-retest}$ = 0.92 | | | | |
| Total sample of Ss all test positions combined N = 400 (retest data not included) | 4.0 | 8.2 | 15.0 | 11.0 |

data for the total sample) The test retest correlation was +0.92 a respectable reliability coefficient (See Table 10 ) While there was no significant improvement in median performance on the test (nonparametric *t* test) there was some reduction in the range of performance on the retest between the 10th and 90th percentile this reduction in inter S variability is even more marked when the performance of the entire sample of Ss is considered Thus it can be seen in Table 10 that in the retest as well as in tests on the MFD test which followed other types of discrimination tests the point separating the poorest 10% of the Ss from the remaining Ss (90th percentile) was nearer the average performance than when the test was presented as the very first of the battery It would appear that both experience on the specific test, as well as experience on other types of discrimination tests while not affecting the performance of the average and better than average Ss did improve performance of the Ss with the poorest monotic frequency discrimination This differential improvement poses a problem for anyone concerned with the establishment of ranges of normal performance for diagnostic purposes since it points to the possibility that practice—and quite probably motivation—may enter in a significant fashion into the performance of presumably normal Ss to extend the upper limits of the normal range Such an extension of course decreases the effectiveness of the testing instrument in distinguishing between normal and pathological functioning

Another question of importance is the relationship between performance on the present test and thresholds reported in other studies A summary of some of the studies is presented in Table 11 The information in the table represents data (DLs) derived from investigations that utilized a number of different psychophysical methods and various modes of stimulus presentation The studies which were selected were those most amenable for comparison with our study i e group data the majority of which were collected from relatively large samples of naive Ss

TABLE 11 *Summary of frequency discrimination studies*

| Investigator | Ref year | SL (dB) | SS | Type of stimulus | Psychophysical method | Value (cps) |
|---|---|---|---|---|---|---|
| Shower & Biddulph | 1931 | 40 | 5 experienced | Modulated frequency (MF) | Limits | 3.6 (mean) |
| Turnbull | 1944 | 60 | 6 experienced | Separate stimuli (SS) | Constant stimuli (CS) | 2.80 (mean) |
| Harris (*a*) | 1948 | 50 | 54 inexp | SS | Modified CS variable progressively closer to standard | 4.8 (mean) |
| Harris (*b*) | 1948 | 60 | 24 inexp | | Modified CS random variable | 4.7 (mean) |
| Harris | 1952 | 30 | 60 inexp | SS | AX | 3.61 (median) |
| Meurman | 1954 | 20 | 36 inexp | MF | Modified limits (increasing) | 6.7 (mean) |
| *Present study* | | 40 | 400 inexp | SS | Modified constant stimuli | 8.2 (median) |

*All of the studies* show *lower* average DLs than that obtained *in* our present study. Meurman's DL value is closest to the DL of 8.2 from our sample. The results *from* the various investigations *may* reflect either *procedural or S differences. In the present study, we believe they reflect S* differences primarily rather than procedural differences since in a pilot study well practiced Ss did *as* well *on* the present test as on tests utilizing other psychophysical methods. It would *appear* in large part then, that the poorer *performance* obtained in the present study reflects the fact that (1) the Ss were *not* well practiced, (2) some of the Ss were not highly motivated and (3) the Ss were *unselected* with respect to their discrimination *ability*. In many psychophysical studies, of course, these factors have been *quite properly* controlled in order to obtain thresholds of minimum size and maximum reliability. However we would raise the question of the *representativeness* of those data and (1) whether they could be used to *predict* performance of unselected and unpracticed Ss or (2) whether they could, for example, be used as normative bases against which to *compare* patients tested in the clinic.

## DICHOTIC FREQUENCY DISCRIMINATION (DFD)

*In this test (DFD) pulses were presented simultaneously to both ears* 40 dB above each ear's threshold. Standard pulses were always presented to the left ear, while standard and *comparison tones* were presented to the right ear (Table 7). The S's task therefore was to determine which train of pulses contained the higher frequency comparison tones to the right ear. *Phenomenologically*, at supra threshold levels the tone pulses

TABLE 12 *Mean test scores and standard deviations for dichotic frequency discrimination test for each test position*

| Test position | Mean (cps) | Standard deviation (cps) |
|---|---|---|
| 1 | 4 27 | 4 5 |
| 2 | 6 19 | 9 8 |
| 3 | 4 91 | 5 9 |
| 4 | 4 93 | 9 5 |
| 5 | 3 98 | 4 7 |

appeared as single images in the median plane of the head when both ears received standard pulses and as separate tones, located in each ear, when the comparison tones were presented to the right ear In the latter case, pitch differences could also be detected in the two ears if the frequency differences were well above threshold, however, such pitch differences were much less obvious than the splitting of the image and, near threshold, only the splitting of the image was reported

## RESULTS

An analysis of variance to determine whether there was a significant test position effect was carried out It was found not to be significant ($F = 1\ 09$, $df$ 4,395) and scores of all Ss were combined Mean test scores and SDs for the various test positions are shown in Table 12

The distribution of dichotic frequency discrimination thresholds for all Ss is shown in Fig 2, percentile equivalents are presented in Table 13 Here again, the distribution was markedly skewed—in fact, it is J-shaped with the modal threshold 0 0 cps difference

TABLE 13 *Percentile equivalents of scores on the dichotic frequency difference test* ($N = 400$)

| Percentile (%) | Dichotic diff thr (cps) | Dichotic diff thr (relative) | Percentile (%) | Dichotic diff thr (cps) | Dichotic diff thr (relative) |
|---|---|---|---|---|---|
| 5 | 00 | 00000 | 55 | 3 37 | 00337 |
| 10 | 01 | 00001 | 60 | 3 87 | 00387 |
| 15 | 04 | 00004 | 65 | 4 30 | 00430 |
| 20 | 24 | 00024 | 70 | 5 60 | 00560 |
| 25 | 66 | 00066 | 75 | 6 47 | 00647 |
| 30 | 97 | 00097 | 80 | 7 80 | 00780 |
| 35 | 1 20 | 00120 | 85 | 9 30 | 00930 |
| 40 | 1 50 | 00150 | 90 | 12 00 | 01200 |
| 45 | 1 98 | 00198 | 95 | 15 00 | 01500 |
| 50 | 2 80 | 00280 | 100 | 80 00 | 08000 |

Table 14 *Summary of performance on the dichotic frequency discrimination test*

| | 10th percentile (cps) | 50th percentile (cps) | 90th percentile (cps) | Diff (90th 10th) (cps) |
|---|---|---|---|---|
| Test presented in first position of the battery sequence N = 80 | 0.0 | 3.0 | 10.0 | 10.0 |
| Retest immediately following first presentation N = 80 $r_{test\ retest} = 0.80$ | 0.0 | 1.6 | 9.0 | 9.0 |
| Total sample of Ss all test positions combined N = 400 (retest data not included) | 0.1 | 2.8 | 12.0 | 11.9 |

For the 80 Ss who took this as the first test of the battery it was repeated immediately to determine test reliability and the effect of practice The test retest correlation was 0.80 and represents a reasonable degree of reliability However in contrast to the test retest results with the MFD Test (in which there was no significant improvement of the performance but a reduction in the 10th 90th percentile range) the retest data for the DFD Test show a decided improvement ($p = 0.01$) in median performance with little change in the range of performance (see Table 14) It will also be noted that the median performance for the total sample of Ss, in which test position was systematically varied did not differ in any marked manner from the median for test position 1 Thus while there is evidence of improvement in performance as a result of practice on the specific test there is no evidence of change as a result of experience on other discrimination tests

One of the most striking aspects of performance on the DFD Test is the large number of Ss who detected zero differences in frequency between the comparison tone in the right ear and the standard tone in the left ear It is clear that since pitch was not a cue phase relationships between the tones to each ear were utilized In considering the results it is important to note the manner in which the standard and comparison tones were generated When standard tones were presented to both ears they were generated by the same oscillator and were always in phase Comparison tones of course were generated by a second oscillator Consequently even with both oscillators set to the same frequency there were phase differences which were utilized by those Ss able to detect the comparison tones when set to 1000 cps Further even though both oscillators were set to the same frequency to the nearest tenth of a cycle a study of the tones revealed gradual shifts in relative phase angles during each [illegible] msec pulse Since such shifts were slow and the pulse length short it appears probable that the principle cue utilized by the Ss for

the detecting zero frequency differences was the absolute phase difference rather than the changing phase angle relationships However, when the comparison tone was set to frequencies above the 1000 cps standard tone it is quite possible that the shifting relative phase angles as well as pitch differences served as additional cues

A comparison of the monotic and dichotic thresholds reveals striking differences in their sizes The median monotic differences threshold was 8 9 cps while the median dichotic threshold was 2 8 cps From this it is quite clear that the additional cues provided by the phase relationships of the two tones was utilized by many of the Ss The correlation between performance on the MFD and DFD Tests was computed and was found to be surprisingly low—only +0 21 (corrected for attenuation $r$ 0 24) It is clear from this that quite different cues were utilized by the Ss in detecting the presence of the comparison tones in the right ear in the two tests and that the ability to utilize the phase cues was but poorly related to the ability to detect successive pitch differences even though both were related to frequency differences

# INTENSITY DISCRIMINATION

Intensity difference thresholds were determined for both monotic and dichotic conditions The data for each condition will be presented separately and the findings then compared

## Monotic Intensity Discrimination

A detailed description of the forced-choice method used in determining these thresholds can be found in the Procedure section Intensity differences between standard and comparison tones for the various blocks of trials are indicated in Table 7 The intensity differences used were based on data from a pilot study and proved to be quite adequate, since less than 1% of the participating students had thresholds that exceeded the largest difference and had to return for retesting

### Results

An analysis of variance was performed to determine if there was a significant test position effect It was found not to be significant ($F = 1\ 12$, *df* 4,395) and scores for all Ss were combined Mean test scores and SDs for each test position are shown in Table 15

The distribution of thresholds for all Ss is shown in Fig 3, percentile equivalents are presented in Table 16 Here again, the distribution was positively skewed

For the 80 Ss who were given the test first the reliability of the MID test was assessed by repeating it immediately The test-retest coefficient ($N = 80$) was 0 62, not a particularly high reliability, and much lower than

Table 15 *The means and standard deviations for the monotic intensity difference threshold Each mean is based on an N of 80*

| Test position | Mean (dB) | Standard deviation (dB) |
|---|---|---|
| 1 | 2 59 | 1 43 |
| 2 | 2 86 | 1 91 |
| 3 | 2 80 | 1 72 |
| 4 | 2 38 | 1 29 |
| 5 | 2 77 | 1 75 |

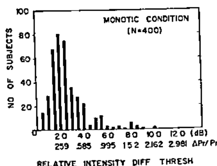



Fig 3

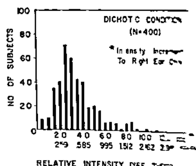



Fig 4

Fig 3 Frequency distribution of monotic relative intensity difference thresh…

Fig 4 Frequency distribution of dichotic relative intensity difference thresh…

those found for the frequency discrimination tests Also of inte… the effect of practice on intensity discrimination The retest data … that the median performance decreased by 0 3 dB (Table 17), an … ment which was significant at better than the 1% level In additi… lowering of the median threshold, the 10th and 90th percentile … decreased indicating that practice was more or less effective … whatever their initial level of discrimination It appears tho… Ss with the poorer ability profited more from practice for th… of the range was curtailed by 0 40 dB

Data of other investigators are provided in Table 18 so that … can be made with our results Our total sample yielded a … ference threshold of 2 27 dB which is almost twice that … studies at the same frequency and SL Loeb and Hawkes' …

Table 16 *Percentile equivalents of scores on the mon… intensity difference tests* (N = 400)

| Percentile (%) | Intensity diff thr (dB) monotic | dichotic (a) | dichotic (b) | Percentile (%) | Inter… monotic |
|---|---|---|---|---|---|
| 5 | .97 | 1 25 | 0 67 | 55 | [illegible] |
| 10 | 1 00 | 1 30 | 0 82 | 60 | [illegible] |
| 15 | 1 36 | 1 78 | 0 98 | 65 | [illegible] |
| 20 | 1 53 | 1 98 | 1 10 | 70 | [illegible] |
| 25 | 1 68 | 2 27 | 1 28 | 75 | [illegible] |
| 30 | 1 77 | 2 45 | 1 40 | 80 | [illegible] |
| 35 | 1 95 | 2 56 | 1 47 | 85 | |
| 40 | 2 00 | 2 71 | 1 57 | 90 | |
| 45 | 2 16 | 2 95 | 1 73 | 95 | |
| 50 | 2 27 | 2 99 | 1 74 | 100 | |

*Note* See text for description of manner in which …

TABLE 17. *Summary of performance on the monotic intensity discrimination test on initial test, retest, and for all test positions combined*

| | 10th percentile (dB) | 50th percentile (dB) | 90th percentile (dB) | Diff (90th–10th) (dB) |
|---|---|---|---|---|
| Test presented in first position of the battery sequence, N = 80 | 1 10 | 2 20 | 4 25 | 3 15 |
| Retest immediately following first presentation, N = 80 | 1 00 | 1 90 | 3 75 | 2 75 |
| $r_{test\ retest}$ = 0 62 | | | | |
| Total sample of Ss, all test positions combined N = 400 (retest data not included) | 1 00 | 2 27 | 4 97 | 3 97 |

furnished the closest comparison with respect to procedure and our median is about 1 27 dB greater than their mean Their data were collected at 30 and 60 dB SL, but as there is only 0 1 dB difference between the two DLs at those levels, we may assume that the value for 40 dB SL was about 1 0 dB Since the Loeb and Hawkes sample consisted of six staff members, who were practiced in auditory tasks, the differences in DL most probably reflects S difference due to experience Our retest data showed a significant lowering of the median DL for 80 Ss (2 20 dB to 1 90 dB)

Harris reports a median DL of approximately 1 0 for 21 inexperienced Ss in a forced choice procedure comparing a standard and a variable pair of pulses However, he reports that all Ss who did not yield "satisfactory" DLs were retested until the limen became "satisfactory" Harris offers no criterion of "satisfaction", but it appears safe to assume that many of the Ss were no longer inexperienced when final DLs were measured Table 18 also includes data obtained with amplitude modulation and quantal or incremental procedures for a gross comparison For a thorough discussion of relationships among these studies, the reader is referred to Harris' monograph (1963)

## DICHOTIC INTENSITY DISCRIMINATION

The patterning of pulses, which was used in determining the dichotic difference threshold can be seen in Table 7 It will be noted that the left ear received only trains containing standard pulses while the right ear simultaneously received either standard or comparison stimuli As before, the S had to decide whether the first or second train had the pulses that were different" The comparison-standard tone intensity differences in the right ear were the same as used in the monotic test, namely 10, 7, 5,

TABLE 18 *A summary of monotic intensity difference thresholds obtained at IK by other investigators*

| vestigator | Ref year | SL (dB) | Ss | Type of stimulus | Psychophysical method | Value (dB) |
|---|---|---|---|---|---|---|
| esz | 1928 | 40 | 12 experienced | Amplitude modulation (AM) | Limits | 0 8 (mea |
| errick | 1959 | 40 | 3 experienced | Separate stimuli (SS) | Limits | 0 88 (mea |
| | | | | AM | | 0 2 (mea |
| eb & Hawkes[a] | 1967 | 30 | 6 experienced | SS | Limits | 1 1 (mea |
| | | 60 | | | | 1 0 (med |
| arris (a) | 1963 | 40 | 7 experienced | SS | Modified adjustment | 1 57 (mea |
| arris (b) | 1963 | 40 | 6 experienced | AM (replication of Riesz) | Limits | 0 83 (mea |
| arris (c) | 1963 | 40 | 6 experienced | AM (quantal) | Modified adjustment | 1 0 (mea |
| arris (d) | 1963 | 40 | 21 inexp[b] | SS | Modified constant stimuli | 1 0 (med |
| esent study | | 40 | 400 inexp[b] | SS | Modified constant stimuli | 2 27 (med |

[a] Eight pulses alternating between standard and variable ( B condition)
[b] Erratic Ss (or unsatisfactory) called back No criterion for "satisfactory" Ss offered

3 2 and 1 dB With the intensity differences occurring only in one ear, differences between standard and comparison tones can be specified in the two ways the difference in intensity of the tones to the right ear only (*a* Table 16) or the total difference (power) of the signals to both ears (*b* Table 16) Unless indicated otherwise the first definition will be used, however, since the values differ both are presented in Table 16 The range of stimulus differences proved quite satisfactory since only four Ss had to return for retesting because their difference thresholds exceeded the upper limit of the test

The analysis of variance performed to determine if experience with the other discrimination tests would influence behavior on this particular test yielded an $F$ value of less than 1 00 (*df* 4 395) For means and standard deviation see Table 19 consequently the data for the entire sample was combined and the frequency distribution of the thresholds is presented in Fig 4 the percentile equivalents are shown in Table 16 The median threshold for the entire sample was 2 99 dB with a range from 1 50 (10th percentile) to 5 69 dB (90th percentile) The reliability of the DID test was calculated as with the other tests on test retest data The reliability coefficient based on 80 Ss was 0 62 indicating only a moderate degree of stability

TABLE 19 *The means and standard deviations for the dichotic intensity difference threshold Each mean is based on an $N = 80$*

| Test position | Mean (dB) | Standard deviation (dB) |
|---|---|---|
| 1 | 3.39 | 1.66 |
| 2 | 3.52 | 1.95 |
| 3 | 3.24 | 1.60 |
| 4 | 3.45 | 1.85 |
| 5 | 3.45 | 1.90 |

An examination of the effects of practice on both the discrimination task and the method indicate that the overall performance on the dichotic test closely paralleled that of the monotic presentation The median dichotic threshold upon retesting decreased by 0.30 dB (Table 20), and is statistically significant ($p < 0.01$) The 10th and 90th percentiles, upon retesting, not only had lower values but the range between them was reduced by 0.25 dB Practice evidently improved the performance for most of the Ss and in particular for those who performed most poorly initially As was the case with monotic discrimination no significant threshold change resulted from practice with the method itself for a comparison of performance in the "first position" and for the total sample shows them to be very similar Additionally, the range for the total sample was not narrowed

COMPARISON OF MID AND DID THRESHOLDS

MID and DID thresholds were found to have a moderate correlation of 0.46 (corrected for attenuation, $r = 0.74$) If the absolute values of the monotic and dichotic thresholds are ignored, it will be seen that the two

TABLE 20 *Summary of performance on the dichotic intensity discrimination test on initial test retest and for all test positions combined*

| | 10th percentile (dB) | 50th percentile (dB) | 90th percentile (dB) | Diff (90th 10th) (dB) |
|---|---|---|---|---|
| Test presented in first position of the battery sequence $N = 80$ | 1.80 | 3.00 | 5.45 | 3.65 |
| Retest given immediately following first presentation $N = 80$ $r_{test-retest}$ 0.62 | 1.50 | 2.70 | 4.90 | 3.40 |
| Total sample of Ss all test positions combined $N = 400$ (retest data not included) | 1.50 | 2.99 | 5.69 | 4.19 |

sets of data are quite similar. Neither showed the influence of experience with the procedure *per se*, but both showed quite conclusively the influence of practice with the particular test. The test-retest reliability for each of the intensity tests was identical, namely, 0.62. A comparison of the distributions for the test taken in first position ($N=80$) and the total sample ($N=400$) shows that average performance did not change, but that the larger sample has increased inter-S variability in both instances.

Since both tests involved detection of intensity differences, performance on them should be fairly highly correlated; however, since the louder variable tone was presented only to the right ear, localization cues might conceivably enter in to assist in discriminating differences in the dichotic test. However, this does not appear very likely since the improvement in discrimination on the dichotic test is not as great as that reported by Upton and Holway (1937) and by Churcher *et al.* (1934) in which monotic and dichotic thresholds were compared. Column *b* in Table 16 shows the DID thresholds specified in terms of total difference (both ears combined) in which the increments are specified in terms of power changes; the thresholds are smaller than those found for the monotic test. The nature of the relationship is illustrated in Fig. 5 in which the data points are the power ratio equivalents of the percentile values presented in Table 16 (monotic vs. column *b*). It will be noted that the data are fitted quite adequately by a linear function (correlation between obtained and predicted values = 0.998).

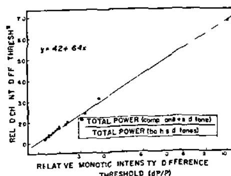



FIG. 5. Comparison of monotic and dichotic intensity difference thresholds; abscissa and ordinate values represent power ratio equivalents of the percentile values shown in Table 16.

The monotic difference threshold for tonal duration was obtained using a standard pulse length of 250 msec; variable pulse lengths ranged from 350 to 260 msec (Table 7) these values having been established in a pilot study which indicated that they would cover the difference thresholds for most of the Ss As it turned out, these stimulus values proved to be sufficient for all but 3% of the sample which had to be retested later (In all such cases Ss exceeded the upper limit of the variable stimuli)

Just as the other discrimination tasks occupied one of the five possible positions in the test battery, so did the duration difference test The means and standard deviations for each of the five positions can be seen in Table 21 A simple analysis of variance performed to determine if test position was a determinant in the obtained thresholds yielded an $F$-value of less than one (*df* 4,395), consequently, the data for all Ss were combined The distribution of these thresholds can be seen in Fig 6 and percentile equivalents in Table 22 The median threshold for the entire sample was 40 msec and the range was 56 msec (10th to 90th percentiles) The test-retest reliability of the MDD test was found to be 0 64, indicating only a moderate degree of stability (Table 23)

An examination of our test-retest data showed that upon retesting, the median performance decreased by 1 msec while the range (10th to 90th percentiles) decreased by 6 msec entirely as a result of the reduction in the 90th percentile thresholds The decrease in the median was not significant when tested statistically and even though the decrease at the upper end of the distribution of scores may conceivably indicate some improvement in performance, it is quite small and reflects very little, if any, practice effect

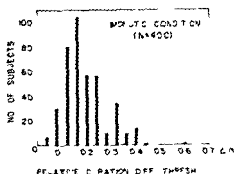



Fig 6 Frequency distribution of monotic relative duration difference thresholds

TABLE 21 *The means and standard deviations for the monotic duration difference threshold Each mean is based on an N = 80*

| Test position | Mean (msec) | Standard deviation (msec) |
|---|---|---|
| 1 | 51 | 21 1 |
| 2 | 47 | 21 7 |
| 3 | 52 | 24 1 |
| 4 | 43 | 17 9 |
| 5 | 42 | 18 6 |

That practice with the method *per se* did not facilitate performance on the duration discrimination task is supported first, of course, by the nonsignificance of the analysis of variance reported above Secondly, the amount of inter S variability did not decrease, the range for the "first position" and the total sample differ by only 1 msec Consequently, the 5 msec difference in the medians probably does not represent any real change in performance

It is not possible to compare directly the data obtained in this study with data collected by other investigators since methods, stimulus parameters, and experience of the Ss varied from study to study Assuming that the frequency and intensity of the auditory stimulus does not affect the duration difference threshold and that the Weber fraction is relatively constant, it is possible, even though no study used the present combination of stimulus parameters, to estimate the Weber fraction for a standard stimulus having a duration of 250 msec from other studies Going back about 100 years to the time of Vierordt's classic study (1868), we find

TABLE 22 *Percentile equivalents of scores on the monotic duration difference test (N = 400)*

| Percentile (%) | Diff thr (msec) | Diff thr (relative) | Percentile (%) | Diff thr (msec) | Diff thr (relative) |
|---|---|---|---|---|---|
| 5 | 20 | 08 | 55 | 44 | 18 |
| 10 | 25 | 10 | 60 | 48 | 19 |
| 15 | 28 | 11 | 65 | 48 | 22 |
| 20 | 31 | 12 | 70 | 54 | 22 |
| 25 | 33 | 13 | 75 | 55 | 22 |
| 30 | 35 | 14 | 80 | 62 | 25 |
| 35 | 37 | 15 | 85 | 70 | 28 |
| 40 | 40 | 16 | 90 | 80 | 32 |
| 45 | 40 | 16 | 95 | 91 | .36 |
| 50 | 40 | 16 | 100 | 150 | 60 |

3* — 66°966

TABLE 23 *Comparison of percentile equivalents for the monotic duration difference thresholds*

| | 10th percentile (msec) | 50th percentile (msec) | 90th percentile (msec) | Diff (90th–10th) (msec) |
|---|---|---|---|---|
| Test presented in first position of the battery sequence N = 80 | 25 | 45 | 81 | 56 |
| Retest immediately following first presentation, N = 80 $r_{test\ retest} = +0.64$ | 25 | 44 | 75 | 50 |
| Total sample of Ss, all test positions combined, N = 400 | 25 | 40 | 80 | 55 |

that the Weber fraction was 0.18, or 45 msec for a standard of 250 msec. More recently, Small and Campbell using a forced-choice procedure involving a more difficult type of judgment than the present study also obtained a relative difference threshold of 0.18 (by our estimate) (1962). Other studies have reported thresholds of the same approximate size Henry, 0.19 (1939), Stott, 0.14 (1955), Creelman, 0.14 (1961) The data agree well with those obtained in this study (Table 22)

Evidently, in the determination of the difference threshold for tonal duration, the degree of experience, method utilized, and practice with the discrimination itself make little difference in the obtained values

A measure of speech discrimination was provided by the administration of the Rhyme Test described earlier The five parallel forms of 50 words each were used (Fairbanks, 1958) In this study, all five forms were presented consecutively so that the Ss had to complete a total of 250 words In order to vary the level of performance on the test for each S, the words were mixed with broad-band noise of varying levels when duplicated on the FM recorder, the upper limit of the noise was determined by the tape recorder's upper limit, which was about 6000 cps (see Appendix A) The

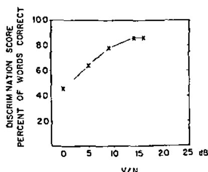



Fig 7

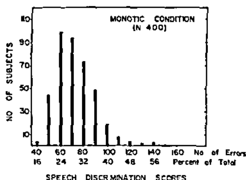



Fig 8

Fig 7 Mean articulation curve

Fig 8 Frequency distribution of monotic speech discrimination scores represent number of errors on total Rhyme Test

Table 24 *Percentile equivalents of total error scores on speech discrimination test* ($N = 400$)

| Percentile (%) | Total errors | Per cent of total | Percentile (%) | Total errors | Per cent of total |
|---|---|---|---|---|---|
| 5 | 50 | 20 0 | 55 | 72 | 28 8 |
| 10 | 53 | 21 2 | 60 | 75 | 30 0 |
| 15 | 56 | 22 4 | 65 | 77 | 30 8 |
| 20 | 58 | 23 2 | 70 | 79 | 31 6 |
| 25 | 60 | 24 0 | 75 | 82 | 32 8 |
| 30 | 62 | 24 8 | 80 | 86 | 34 4 |
| 35 | 64 | 25 6 | 85 | 89 | 35 6 |
| 40 | 66 | 26 4 | 90 | 94 | 37 6 |
| 45 | 68 | 27 2 | 95 | 102 | 40 8 |
| 50 | 70 | 28 0 | 100 | 153 | 61 2 |

TABLE 25 *Summary of performance on five forms of rhyme test (error scores).*

| Rhyme test form | Test position | V/N | 10th percentile Errors | 10th percentile % | 50th percentile Errors | 50th percentile % | 90th percentile Errors | 90th percentile % | Diff (90th–10th) Errors |
|---|---|---|---|---|---|---|---|---|---|
| RT 5 | 1 | 16 | 3 | 6 | 7 | 14 | 13 | 26 | 10 |
| RT 3 | 2 | 14 | 4 | 8 | 7 | 14 | 13 | 26 | 9 |
| RT 2 | 3 | 9 | 7 | 14 | 11 | 22 | 17 | 34 | 10 |
| RT 4 | 4 | 5 | 13 | 26 | 18 | 36 | 24 | 48 | 11 |
| RT 1 | 5 | 0 | 22 | 44 | 27 | 54 | 33 | 66 | 11 |

V/N ratio decreased on each successive test form so that S's performance (percent of words correctly identified) became poorer The mean articulation curve is presented in Fig 7. However, for purposes of further analysis, the total number of errors will be considered, thus, as with the pure tone tests, low scores represent relatively good discrimination

## RESULTS

The frequency distribution for the total discrimination test was positively skewed, the median score was 70 errors This distribution can be seen in Fig 8 and the percentile equivalents in Table 24, performance at the various V/N ratios is indicated in Table 25 Since administration of the speech test a second time was not possible, split-half scores were calculated by alternately summing the errors on five words at a time The Spearman-Brown (1910) split-half reliability was 0 89—a satisfactory degree of reliability

# AUDITORY AND NON-AUDITORY VARIABLE INTERRELATIONSHIPS

The primary purpose of the present study was to obtain ranges of normal performance on the various discrimination tests The secondary purpose was to determine the extent to which variations in performance on each of the auditory tests were related to each other and to certain non auditory characteristics of the Ss Such information, it was thought,

TABLE 26 *List of non-auditory and auditory variables included in the correlational analysis*

| Variable | Nature of score |
|---|---|
| *Non-auditory* | |
| Sex | Code 0 – male subject 1 female subject |
| Race | Code 0 = non Negro subject 1 Negro subject |
| Home language | Percentage of time non English was spoken in home (5 categories) |
| Parents occupation | Fathers level (or major wage earner s) occupational level (0 for unemployed 7 for professional) |
| Parents education | Total number years of both parents formal education |
| Musical training | Total number years of extra school musical training |
| SRA[a] | Total test score (higher score represented greater educational ability) |
| STEP Reading[a] | Total test score (higher score represented greater reading ability) |
| SCAT V[a] | Total test score (higher score represented greater verbal aptitude) |
| SCAT Q[a] | Total test score (higher score represented greater quantitative aptitude) |
| Vocabulary[b] | Total number of incorrect definitions |
| Word fluency[b] | Arbitrary value less total number of words starting with specified letters written in six minutes |
| *Auditory* | |
| Right ear thr of aud | SPL of threshold of audition |
| Left ear thr of aud | SPL of threshold of audition |
| Diff abs thr | Right ear threshold left ear threshold |
| M dur diff thr | Size of diff thr in msec |
| D freq diff thr | Size of diff thr in cps |
| M freq diff thr | Size of diff thr in cps |
| M int diff thr | Size of thr in dB |
| D int diff thr | Size of thr in dB |
| Rhyme test | Total words missed on all five forms of test |
| Rhyme test diff | (Words missed on last two forms) less (Words missed on first two forms) |

[a] See Table 1

[b] Tests described in Appendix B

TABLE 27 *Correlational matrix amon*

| | Sex | Race | Home Lang | Parents Occ | Parents Educ | Musical Train | SRA* | Step Read ** | Seat V ** |
|---|---|---|---|---|---|---|---|---|---|
| Race | 09 | | | | | | | | |
| Home Language | 10 | − 21 | | | | | | | |
| Parents Occupation | 03 | 38 | 01 | | | | | | |
| Parents Education | 04 | 21 | 21 | 58 | | | | | |
| Musical Training | 06 | 04 | 06 | 18 | 20 | | | | |
| SRA* | 03 | 15 | 06 | 24 | 44 | 02 | | | |
| Step Read ** | 03 | 41 | 05 | 32 | 27 | 17 | | | |
| Seat V ** | 01 | 42 | 03 | 39 | 36 | 17 | | 84 | |
| Seat Q ** | 07 | 32 | 08 | 30 | 23 | 18 | | 72 | [illegible] |
| Vocabulary | 00 | 40 | 06 | 38 | 43 | 13 | 64 | 70 | [illegible] |
| Word Fluency | 08 | 25 | 06 | 23 | 23 | 11 | 41 | 47 | − 45 |
| Rt Ear Ab Thr | 06 | 11 | 06 | 01 | 00 | 01 | 07 | 12 | − 12 |
| Lft Ear Ab Thr | 03 | 10 | 00 | 02 | 05 | 01 | 06 | 06 | 09 |
| Diff in Ab Thr | 03 | 00 | 08 | 04 | 07 | 00 | 15 | 07 | − 04 |
| Monotic Duration Diff Thr | 01 | 31 | 04 | 14 | 13 | 09 | 17 | 03 | − 34 |
| Dichotic Freq Diff Thr | 02 | 11 | 00 | 18 | 16 | 09 | 04 | 27 | 31 |
| Monotic Freq Diff Thr | 08 | 06 | 03 | 06 | 06 | 10 | 23 | 21 | 22 |
| Monotic Int Diff Thr | 05 | 14 | 02 | 17 | 09 | 03 | 14 | 19 | 23 |
| Dichotic Int Diff Thr | 04 | 14 | 11 | − 17 | − 11 | − 10 | 01 | − 22 | 21 |
| Rhyme Test | 10 | 18 | 10 | 10 | 19 | 06 | 28 | 32 | 33 |
| Rhyme Test Diff | 07 | 10 | 01 | 01 | 05 | 05 | 12 | 11 | 10 |

*Significance Levels*

Based on total sample ($N = 400$)
$r = 0.10 \quad p < .05 \qquad r = 0.13 \quad p < .01$

Based on Catholic school students ($N = 104$)
$r = 0.20 \quad p < .05 \qquad r = 0.25 \quad p < .01$

Based on public school students ($N = 296$)
$r = 0.11 \quad p < .05 \qquad r = 0.15 \quad p < .01$

would be useful in developing an understanding of some of the sources of test performance variation Certainly, such diverse factors as motivation and musical training might be expected to be related to auditory discrimination and these in turn might well be related to factors such as home environment intelligence achievement in school etc On the other hand it was not unreasonable to expect a generalized auditory discrimination

*uditory and non-auditory variables*

| | Vocabulary | Word Fluency | Rt Ear Ab Thr | Lft Ear Ab Thr | Diff in Ab Thr | M D Thr | D I Thr | M F Thr | M I Thr | D I Thr | Rhyme | Mean | S D |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | 21.6 | 5.8 |
| | | | | | | | | | | | | 1.3 | 2.4 |
| | | | | | | | | | | | | 110.0 | 12.9 |
| | | | | | | | | | | | | 287.0 | [illegible] |
| | | | | | | | | | | | | 276.0 | 15.3 |
| | | | | | | | | | | | | 291.0 | 16.4 |
| | | | | | | | | | | | | 19.0 | 6.1 |
| | 52 | | | | | | | | | | | 34.3 | 7.8 |
| | 10 | 08 | | | | | | | | | | 9.1 | 1.3 |
| | 10 | 07 | − 64 | | | | | | | | | 9.1 | 1.5 |
| | 00 | 00 | 39 | − 46 | | | | | | | | 0 | 3.8 |
| | 29 | 29 | 00 | 05 | − 06 | | | | | | | 47.0 | 21.0 |
| | 25 | 26 | 10 | 04 | 07 | 37 | | | | | | 4.8 | 7.2 |
| | 15 | 18 | − 05 | − 07 | 02 | 21 | 21 | | | | | 10.1 | 11.3 |
| | 13 | 20 | − 15 | − 12 | − 03 | 39 | 25 | 18 | | | | 2.7 | 1.6 |
| | 16 | 18 | − 08 | − 01 | − 08 | 34 | 27 | 21 | 46 | | | 3.4 | 1.8 |
| 3 | 34 | 31 | 33 | − 24 | − 09 | 29 | 25 | 4 | 33 | 27 | | 72.4 | 17.0 |
| | 12 | 10 | 14 | 07 | 09 | 09 | 10 | 12 | 11 | 00 | 19 | 30.2 | [illegible] |

factor—though if found it would not necessarily reflect general differences in the Ss auditory systems

The decision of which non auditory variables to include clearly involved a good deal of guess work since there is little information concerning the extent to which such factors are related to auditory discrimination A case could certainly be made for predicting a moderate relationship between scholastic aptitude test scores and the auditory test scores and so such information were included in the analysis of the data Although there are no strong *a priori* reasons to expect significant relationships between sex and race variables and discrimination test scores they were included since this study was viewed as essentially exploratory in nature and a broad range of variables was consequently desirable Since it appeared probable that motivational factors and experiential backgrounds would be

related to test performance it was decided to obtain certain items of personal data that might reflect such factors Consequently data were obtained on the educational level of the Ss' parents and upon the occupational level of the father as indicants of the socio economic status of their homes Since a verbal discrimination test was to be included information concerning the extent to which English was spoken in the home was obtained in addition paper and pencil tests of vocabulary and word fluency were also administered as further measures of the Ss knowledge of English verbal material Finally the extent of the Ss musical training was determined since this was expected to be related to pure tone discrimination scores

We would not suggest of course that the non auditory factors included in this study would relate more highly to the auditory discrimination scores than many other factors The decisions to include them were of necessity somewhat arbitrary However with the exception of the Ss sex and race about which no predictions were made, we expected to find them related to various discrimination tests in a statistically significant—though possibly not a practically significant manner

The auditory scores studied included all of the discrimination test scores and in addition the Ss thresholds of audition at 1000 cps in both ears as well as the difference in such thresholds between ears this latter measure was included so that marked differences in acuity between the two ears might be considered in relation to the two dichotic discrimination tests Listed in Table 26 are all of the variables included in the correlational analyses In each case the direction of scoring or the nature of the coding is indicated so that the correlation matrix (Table 27) can be correctly interpreted

## Zero Order Correlations

Only correlations involving the auditory tests and reflecting relationships of practical significance —i e 0 30 or greater indication of about 10% or more common variance will be considered

### *Relationships of non auditory variables with discrimination performance*

The non auditory variables can be conveniently divided into two general categories the personal data items and the aptitude and achievement scores obtained on various paper and pencil tests The first category includes the variables of sex race home language parents occupation parents education and musical training of these only one was correlated in a practically significant manner with any auditory test This was the correlation between race and the MDD test (+0 30) the direction of the correlation indicated that the white Ss tended to do better than non white Ss Why this relationship should show up for this single discrimina

TABLE 28 *Mean difference thresholds of students with musical training*

| Discrimination test | Musical training: Less than 3 yrs (N = 75) | 3 yrs or greater (N = 78) |
|---|---|---|
| Monotic duration difference threshold | 46 msec | 43 msec |
| Monotic intensity difference threshold | 2 8 dB | 2 7 dB |
| Dichotic intensity difference threshold | 3 4 dB | 3 1 dB |
| Monotic frequency difference threshold | 10 6 cps | 7 2 cps[a] |
| Dichotic frequency difference threshold | 6 1 cps | 3 2 cps[b] |
| Speech discrimination | 71 errors | 70 errors |

[a] Mean difference $p < 01$
[b] Mean difference $p < 05$

tion test when all others are far lower is puzzling and we tentatively are disregarding it The lack of relationships is not too surprising since the variables were included primarily because the study was an exploratory one, and not because of *a priori* reasons to predict strong relationships It was felt, however, that we should determine whether any significant amount of the sizable individual differences evident on the auditory discrimination tests might be accounted for by such non-auditory characteristics of the Ss

The single personal data variable we did expect to be related to auditory performance was the musical training variable As is evident, however, our expectations proved to be completely wrong This is somewhat puzzling, and in considering this finding in some detail, it was noted that the musical training reported by the students was frequently limited in extent and often terminated well before they were tested (see Table 28) It was therefore decided to select only those Ss with more than three years of musical training and who were actively continuing When these Ss were investigated, it was found that those students who had three years or more musical training did have significantly better discrimination for both of the frequency tests Whether musical training *per se* developed this finer frequency discrimination is of course, not known It is of considerable interest, however that none of the other discrimination tests showed a similar relationship apparently, whatever the basis of the relationship, it is not related to the other discrimination abilities measured in this study

The remaining non auditory variables were test scores obtained on various educational aptitude and achievement tests, and upon tests of vocabulary and word fluency Here the relationships with auditory discrimination performance tended to be higher Among the discrimination tests, the Rhyme Test tended to relate most highly to these paper and pencil tests; in this latter case, correlations ranged from 0 28 to 0 34 (Table 27) For

TABLE 30 *Factor analysis on total sample for auditory variables only limited to four factors Quartimax rotation*

| | *1* | *2* | *3* | *4* |
|---|---|---|---|---|
| Rt Ear Ab Thr | − 02 | 90 | 33 | 07 |
| Lft Ear Ab Thr | 00 | 85 | −.50 | 01 |
| Diff in Ab Thr | 03 | 03 | 99 | 03 |
| Monotic Duration Diff Thr | 71 | 08 | − 06 | 11 |
| Dichotic Frequency Diff Thr | 62 | 17 | 16 | 18 |
| Monotic Frequency Diff Thr | 38 | − 10 | 04 | 41 |
| Monotic Intensity Diff Thr | 74 | − 18 | − 01 | − 10 |
| Dichotic Intensity Diff Thr | 74 | − 05 | − 08 | − 12 |
| Rhyme Test | 45 | − 46 | 14 | 44 |
| Rhyme Test Diff | − 01 | 12 | 06 | 87 |
| Per cent of common variance | 31 | 27 | 21 | 18 |

*Factor 2* quite clearly relates to the threshold of audition, here, as in the earlier factor analysis, Rhyme Test performance is found to be negatively related to this factor We are at a loss for a simple and satisfactory explanation for such an *inverse* relationship between thresholds of audition and verbal discrimination

Factor 3 can be seen to be primarily related to differences in the thresholds of audition for the Ss' two ears It appears that variations between the two ears is not related to either monotic or dichotic discrimination performance.

Factor 4 as was its counterpart Factor 6 in the earlier Factor Analysis is still the puzzler—loading with the MFD Test the Rhyme Test, and the Rhyme Test difference (slope) The two measures of verbal performance are of particular interest, indicating that Ss with poorer overall verbal discrimination performance exhibited the most rapid decrement in discrimination as the noise level was increased Presumably there may be a slight tendency for masking effectiveness to be inversely related to verbal discrimination ability This does not, in retrospect, appear to be too surprising Just exactly how the MFD test crept into the picture, however, is difficult to say and we would prefer to ignore its presence

*Predictions based upon multiple correlations*

Since the Rhyme Test measures verbal recognition, probably the single most important task of the auditory system, it was decided to determine

TABLE 31 *Standard partial regression coefficients for all variables and for auditory variables only, criterion measure is the Rhyme Test score*

| Variables | All variables included (N = 296) $b^*$ | Auditory variables only (N = 400) $b^*$ |
|---|---|---|
| Sex | 00465[a] | |
| Race | 00239 | |
| Language | 00252 | |
| Parents occupation | 00402 | |
| Parents education | 01484 | |
| Musical training | − 00335 | |
| Step Reading | − 10227 | |
| Scat V | 01094 | |
| Scat Q | 06320 | |
| Vocabulary | 06056[a] | |
| Word fluency | 05844[a] | |
| AL R | 02727 | − 07961[a] |
| AL L | 14907 | 21074 |
| AL diff | 10066 | 15614 |
| MD D | 09436 | 17253 |
| DF D | 05685[a] | 06905[a] |
| MF D | 07516[a] | 08958[a] |
| MI D | 01291[a] | 01550[a] |
| DI D | 00769 | 00815 |
| R | 63607 | 52881 |
| Reduced R[2] | 61355 | 51579 |
| $R^2$ | 40458 | 27333 |
| $SE_y$ | 16 7500 | 17 0456 |

[a] $t$ test of significance $p = 0\ 05$

how well such performance could be predicted For this purpose, two multiple regressions were computed, one based upon all of the variables and the other based only upon the auditory variables Multiple correlations of 0 64 and 0 53 respectively were found In Table 31, the data for the multiple correlations are presented together with the reduced multiple correlations indicating estimates of its size when applied to new but similar groups of Ss

Considering first the prediction of Rhyme Test performance when all variables are included it should be noted that although six of the variables are significant the MFD Test contributes slightly more to the predicted score than do vocabulary, word fluency and DFD Test which weight approximately equally MID and sex although significant weight very lightly

When only auditory variables were used in predicting Rhyme Test performance, it can be seen that the MDD Test weights twice that of the MFD, DFD, and AL-R which weight approximately equally. Again although MID is significant, it contributes little to the predicted scores. In general, the variables measured account for little of the total variance on the Rhyme Test—40% and 27% for all variables and auditory variables respectively.

# SUMMARY AND CONCLUSION

The major considerations which led to this study arose from our interest in the usefulness of auditory discrimination tests as indices of auditory-system aberrations and from the recognition that performance on discrimination tests usually reveals considerable variance Since a test, if such variance is too large, can be of little use in the specification of abnormal or pathological conditions, it appeared important that normal ranges of discrimination performance be determined

We chose deliberately to test randomly selected, untrained, and in a few cases apparently non-optimally motivated Ss This was done because the majority of the studies to date have reported on the performance of well trained, well-motivated, and highly-selected Ss and, consequently, have provided very little useful information on what might be considered to be 'normal' discrimination—the term here being used in the context of the development of test norms against which to evaluate the individual performance of a S (or a patient) who could reasonably have been drawn from the norm group's parent population It is clear, of course, that one cannot select a norm group having universal applicability, and the group reported upon here is not one In two particular aspects, this group falls short of being an ideal norm group against which individual patients might be compared The first restriction of age range we judge to be of little importance The second level of motivation, however, may have led to nonrepresentative data, at least, in a limited manner Thus, we presume that the usual patient who has gone to the clinic for testing because of concern with his hearing will be well-motivated to perform at his best when presented with a hearing test we doubt that this was the case with every one of the Ss tested in the present study In other respects however, we believe that the Ss reported upon here do form a reasonably acceptable norm group against which the individual patient's performances can be compared since the usual patient is not well practiced with auditory discrimination tests, nor is he pre-selected because of the adequacy of his discrimination performance On the basis of these considerations, then we would suggest that the levels of discrimination performance reported here may be slightly larger than they should be because of the motivational factor, however, they certainly provide far more meaningful normative ranges than have heretofore been available

In considering the findings, four aspects of the data are of particular interest First, the average level of performance with the exception of the duration test, was significantly poorer than data usually reported We

suggest that this reflects two factors (the relative effects of which are unknown), namely, practice and selective processes by which Ss are chosen With few exceptions, auditory discrimination studies have utilized well-practiced Ss and (while the matter far too often goes unmentioned) presumably only Ss having what E considers to be adequate discrimination ability The poorer average performance found in this study was expected, the size of differences between this and other studies, however, indicate the very significant effect of practice and selection of Ss upon the data We believe that the relative effects of these factors is of considerable importance—and should be explored in detail

The second significant aspect of the data concerns the very sizable ranges of performance on each of the tests This again is not surprising, though it does clearly point up the problem faced by anyone concerned with developing finely focused diagnostic tests In this connection, the obvious question to be raised is the extent to which the ranges of discrimination reflect true ability rather than variations of a non-auditory nature, as for example, the S's understanding of the task, his motivation, or his training on the specific task or similar tasks Further, if one takes the assumption of the normal distribution of abilities at all seriously, then the positively skewed distributions of scores (i e, toward poorer discrimination) obtained on each of the tests suggest possible interactions between auditory discrimination ability and other factors An obvious question (and only one of several which might be mentioned) is whether poorer discriminators are, in addition to their lack of skill, also more poorly motivated What, for example, would have happened if this study had been carried out with conditions arranged so that positive payoffs for correct responses had been utilized? Would one find greater improvement among the poorer discriminators? If one did, would this indicate an interaction or only a skewed distribution of motivation? Clearly, many additional studies on sizable samples of Ss must be carried out before the nature of the distribution of discrimination ability can be determined with any degree of certainty

Another significant aspect of the data is also directly related to testing considerations This is the reliability of test performance One of the most striking findings of the present study was the fact that, despite highly similar test procedures for all of the tests, test-retest reliabilities varied tremendously between the tests, ranging from 0 92 for the MFD Test to 0 62 for the MID and DID, and 0 64 for the MDD Tests Apparently our unpracticed Ss had better-established criteria for judging pitch than for judging loudness or duration Possibly, Steven's distinction between metathetic and prothetic continua (1957) is important here, judgments of substitutive processes may be more stable than judgments of incremental activities (either increments in level of activity, as in the MID and DID Tests, or increments in duration of activity, as in the MDD Test). Certainly, the low reliabilities for the intensity and duration discrimination tests

indicate either the need for an alteration in the test procedure or a lengthening of the test[4]

A fourth aspect of interest is the interrelationships found between performance on the various discrimination tests In general, while a small general discrimination factor appears to exist, it is clear that, with the possible exception of the MID and DID Tests, the various discrimination tests tend to be relatively independent of each other. (We recognize that if the reliabilities on the MID, DID, and MDD Tests had been higher, the degree of intertest dependence would have been raised somewhat ) However, to the extent that a common factor was found, an important question is raised as to whether it reflects a general test-taking ability or is it restricted to auditory discrimination test-taking A study of the two factor analyses (note Factors 1 and 3 of Table 29, and Factor 1 of Table 30) suggests quite strongly that the major portion of the discrimination tests' common variance does not overlap with performance on the various paper-and pencil scholastic tests, consequently, there is no evidence that we have a general test-taking factor of any significance for this battery of tests We would emphasize, however, not the common variance observed among the various discrimination tests, but their independence since far more individual than common variance was evident

In general summary, we would suggest that, while an initial step has been taken to obtain normative data on a battery of discrimination tests, the many questions raised by the data point to a broad gap in our knowledge about how the usual person hears or what the limits of his hearing abilities are Obviously, many more studies involving large and unselected samples of Ss must be completed before we will be in a position to make definitive statements concerning normal auditory abilities

[4] In this connection it should be noted that each S s difference threshold was defined as the 75% level of correct performance, which was determined from only 32 trials (in the event the threshold level fell between successive blocks of 16 trials each) or only 16 trials if the 75% criterion was obtained on a specific block of 16 trials If individual testing had been possible testing could have been restricted to trials near the S s threshold and many more defining trials could have been utilized with an increase in test reliability

# APPENDIX A

For the reasons stated earlier, a means of pre-recording the stimuli was considered highly desirable In considering the various devices available for this purpose, it must be recalled that earlier work had already fairly well defined the magnitudes of the phenomena the present study was attempting to measure As a result, a relatively accurate guess could be made at the size of the smallest discrimination likely to be made by the best S tested Using these approximations and the rule-of-thumb that any measuring device (in this case the stimulus) should be at least an order of magnitude more precise than the tolerance required on the phenomenon being measured, the Es were able to arrive at a set of specifications which the stimuli had to meet

The best a c biased direct recording audio tape recorder is, naturally, ideally suited to the reproduction of speech and music waveforms with respect to such parameters as distortion (less than 1% total harmonic distortion), signal-to-noise ratio (at least a 50 dB dynamic range) and bandwidth (40 to 15,000 cps) With respect to certain other parameters, relatively insignificant in speech and music recording, but highly important to the present study, this type of machine is seriously lacking One type of test administered in the study was an amplitude discrimination test It was expected that some Ss would be capable of perceiving changes in amplitude smaller than 1 dB As it happens, due to inhomogeneities in the manufacture of magnetic tape and the inability to adequately regulate the motion of the tape over the recording and playback heads, even the best of audio tape recorders will demonstrate short-term amplitude fluctuations of as much as 3 dB With the exception of the speech discrimination test the other tests involved frequency or time discrimination Here, it was expected that a few Ss would be capable of making frequency discriminations of less than 1 cps Short term variations in frequency are caused by inaccuracies in the tape and drive mechanisms of the recorder and are called "flutter" and "wow" A good audio tape recorder is only capable of controlling these parameters to within 0 2% or 2 cps for a 1000 cps tone Using this type of recorder, it would clearly not be possible to control the stimulus with sufficient precision for our testing purposes

Because of these considerations, it was decided to investigate the feasibility of using an FM tape recorder While this type of machine uses the same medium (oxide-coated plastic tape), the recording method (and the cost) is quite different Instead of recording the input waveform directly on the magnetic tape as in direct recording a carrier frequency in the

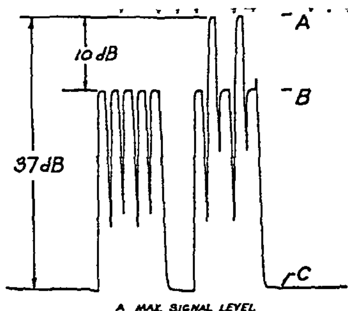



FIG 9 RMS amplitude vs time plot of output of MID test tape made on B & K level recorder A max signal level *B* ref (standard) tone C output noise level

neighborhood of 50 k c is frequency modulated by the incoming waveform and the resulting signal is recorded on the tape The instantaneous value of the recorded signal frequency is directly proportional to the instantaneous amplitude of the input signal As a result upon playback frequency variations not present in the recorded signal but which do appear in the playback due to short term speed variations of the tape become short term frequency and amplitude variations in the output signal (distortion) The motor in the particular machine used was servo-controlled to hold speed variations to an insignificant level Distortion introduced by the discriminator and playback amplifiers brought the over all distortion to around 2% The short term amplitude variation in the output signal however remained at the same low level as specified for speed variation as does the frequency variation The amplitude of the signal coming off the tape is of little significance in this recording process because it is severely limited before detection to eliminate any amplitude variations Although there are available FM tape recorders which more nearly meet the requirements of pure tone discrimination testing their cost is extremely high and they are in general quite large and hard to calibrate By sacrificing some bandwidth and dynamic range the Es were able to obtain a recorder (Honeywell 8100 two-channel) at a reasonable cost which encompassed the following features

(*a*) Bandwidth d c to 6 k c at 15 ips tape speed

(*b*) Dynamic range 40 dB (with proper tape i e 3 M type 959)

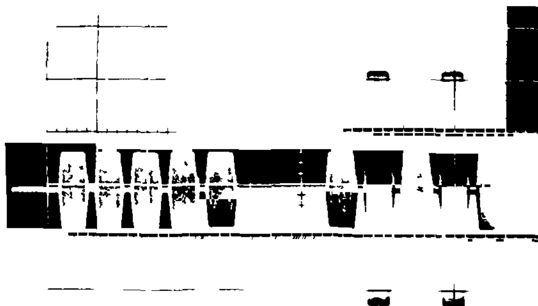


Fig. 10. Oscillograph of pulse train shown in Fig. 9: voltage vs. time; sweep rate 500 msec/cm.

(c) Total harmonic distortion: less than 2%.
(d) Flutter and wow: not measurable with compensation circuit switched in.
(e) Frequency response: ±0.25 dB from d.c. to 5 kc at 15 ips tape speed.
(f) Short term amplitude variations: not measurable.

In addition to ascertaining that the recorder met the manufacturer's published specifications listed above, certain other relevant characteristics were investigated. Figure 9 is an amplitude vs. time plot of one pulse train from our MID test tape. The noise level is seen to be 27 dB down from the standard signal intensity. For the actual testing, this signal-to-noise ratio was further enhanced to 35 dB through the use of a bandpass filter set to 1 kc. Figure 10 is an oscillograph of the same train plotted in Fig. 9.

Two other characteristics of interest were the time stability of the signal and the time relationship between the two channels on the tape. Figure 11 is a one minute exposure of a 1-kc tone. Because the oscilloscope triggers at the same voltage on each sweep, one would expect any frequency changes to manifest themselves as *steadily increasing phase difference* as the sweep progresses, thus widening the trace on the right side. It appears that changes, at least over a 10 msec interval, are of no significant magnitude.

While the outputs of the two channels of the tape recorder differed in time by an amount corresponding to about a 20° phase shift at 1 kc., this could be compensated for by adjustment of the filters in the two channels to change their respective time delays. Figure 12 shows the

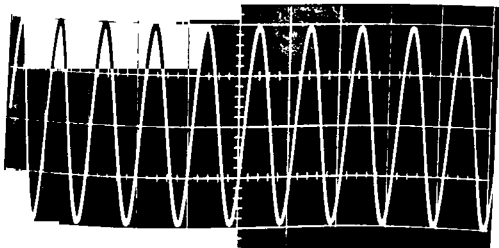
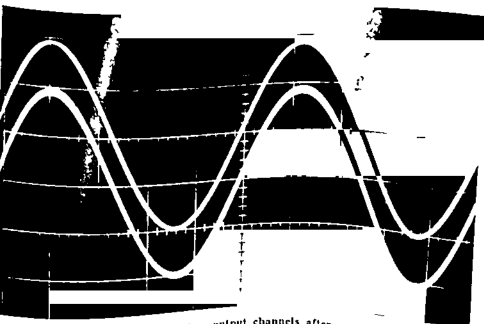


FIG. 11 One minute exposure of repeated 1 ms/cm sweeps displaying the calibration tone at the beginning of a test tape Note the lack of trace widening at the right side of the oscillograph

phase relationship of the signals from the two channels of the recorder after the time-difference compensation

Figure 13 is a 20 second exposure to demonstrate differential phase shifts between the two channels The oscilloscope sweep was locked to the upper trace The phase shifts on successive traces shows as a widening of the



IG 12 Phase relationship of the two output channels after compensating for built in ime difference by adjusting the respective time delays of the filters used in each hannel (single sweep)



Fig. 13 Twenty second exposure of repeated 200 μS/sec sweeps demonstrating the effect of random tape skewing on the phase relationship of the signals from the two output channels

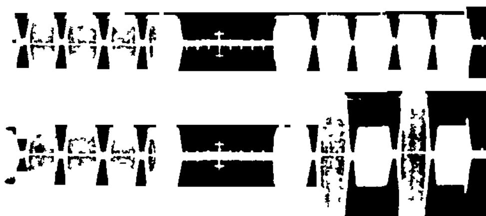


Fig. 14 Both signals for DID test photographed simultaneously on a two beam oscillo[illegible] [illegible]nstrate the simultaneity of the two signals

lower trace It can be seen that variations in the zero crossings vary along the horizontal axis and by approximately $\pm 10°$

Figure 14 demonstrates that the pulses to both ears were presented simultaneously during the dichotic tests

In conclusion, it is felt that the tape recorder produced a stimulus indistinguishable from that produced by the generating equipment, and that once a set of tapes were recorded, the use of the recorder made the testing procedure far easier to administer and much more error free than would have been possible with 'live" equipment

# APPENDIX B

*Vocabulary Test (V-2)*[a]

This is a test of the S's knowledge of word meanings. The format presents a particular word with five synonyms from which the S is to choose the alternative with the same or nearly the same meaning. There are 18 items in each of two parts. Four minutes is allowed for completion of each part.

*Word Endings Test (Fw-1)*[a]

This is a test of the S's ability to think rapidly of as many words as possible that have the same ending. Part 1 has the word ending "ay" while Part 2 has the word ending "ow". The S is allowed to spend three minutes on each of the two parts.

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OTO-LARYNGOLOGICA

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# EXTERNAL CONDUCTIVE HYPACUSIS AND THE FIXED MALLEUS SYNDROME

VICTOR GOODHILL

ACTA OTO-LARYNGOLOGICA · NARVAVÄGEN 16, STOCKHOLM NO

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The 'Fixed Malleus Syndrome' is a term applied either to idiopathic or secondary fixation of the malleus head (caput mallei) which may produce a number of variable oto audiologic sequelae

A rigidly fixed malleal head produces a change in the impedance and in the acoustic energy transfer capacity of the tympanic membrane It is also responsible for similar interference with acoustic energy transfer through the incus The secondary incus effect is due primarily to the nature of the incudomalleal joint (a modified cog type saddle joint) which normally permits incus motion closely geared to that of the malleus In cases where there is true incudomalleal osteoarthritis with obliteration of the joint capsule, this physiologic continuity with the rigidity of the malleus is virtually identical

Fixation of the stapes, either by otosclerotic or pseudo otosclerotic' lesions is one of the most common causes of conductive hypacusis This stapedial fixation is due either to a footplate lesion or, occasionally, to crural fixation and may well be termed 'medial stapedial fixation' A less common cause of stapedial conductive hearing loss is 'lateral stapedial fixation,' which may be defined as the fixating process secondary to immobilization of the incus Such incudal rigidity almost always accompanies, and probably results from, malleal fixation Malleal fixation can thus produce not only immobilization of the tympanic membrane, but in a secondary manner can also produce immobilization of the stapes by lateral displacement towards the oval window in contradistinction to the usual fixation at the vestibular window The stapes thus becomes fixed, not as in otosclerosis or in tympanosclerosis by footplate annular ligamental fusion or by crural promontory fusion but by a restrictive displacement transmitted to the stapedial capitulum from the lateral ossicular chain

Thus, under certain circumstances, one could consider the 'Fixed Malleus Syndrome within the category previously described (Goodhill, 1960) as Pseudo-Otosclerosis,' a term applied to a number of conditions which mimic otosclerosis However, less than complete malleal fixation, accompanied by a relatively mobile incudomalleal or incudostapedial joint, gives a different physiologic end result and hence a different audiologic picture It is incorrect, therefore, to limit the pathologic conception of the "Fixed

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Helmholtz (1874) emphasized the very strong union of the manubrium to the tympanic membrane, particularly at the umbo where the tympanic membrane fibers are especially thickened and blend with fibrocartilaginous tissue which fuses with the manubrial periosteum. At the short process, however, the linkage is looser, forming a sort of incomplete joint fixed only on its two borders.

The second firmest attachment is with the *major tympanic spine*, close to the neck or to the root of the anterior process (processus folianus). The latter, which in children is a long elastic strip of bone extending to the Glaserian fissure, is a short shrunken stump in adults. It runs within the anterior malleal ligament. This stump may or may not join the tympanic spine.

The malleus head (caput mallei) position in the epitympanum is maintained by the *superior ligament*, the *anterior ligament*, and the *lateral ligament*.

The *superior ligament* connects the head of the malleus to the roof of the epitympanum, i.e. to the epitympanic tegmen.

The *anterior ligament* somewhat parallels the course of the *anterior process* (processus folianus), being attached to the neck of the malleus just above the anterior process and extending to the anterior tympanic wall, close to the petrotympanic fissure. Some of its fibers go through this fissure to reach the angular spine of the sphenoid.

The *lateral ligament*, a triangular band of fibers and folds, begins at the posterior aspect of the notch of Rivinus and inserts into the head of the malleus.

In Kirikae's (1960) study of the malleal ligaments, he found much variety in the histological composition. Ligamentous fibers and mucous membrane folds were both represented in variable proportions. In the superior ligament, there was a predominance of folds.

The incus is maintained in position by the *posterior incudal ligament*, connecting the end of its short crus or process to the fossa incudis, and by the somewhat vestigial *superior incudal ligament* (which is hardly more than a fold of mucosa) anchored to the roof of the epitympanum.

The tensor tympani muscle, a rather large structure, sends its tendon to insert into the medial aspect of the malleus. This tendon makes a rather sharp turn 90 degrees around the pulley of the cochleariform process to insert into the manubrium at its superior surface.

The valuable contribution by Proctor (1963*a*) on the "visceral spaces"

Malleus Syndrome" to a subtype within the category of "Pseudo-Otosclerosis."

Thus, while an air-bone gap could conceivably be the only audiologic feature of a completely fixed malleus, in sub-total fixation of the malleus (which might best be termed *increased stiffness of the malleal head*) with a relatively loose incudomalleal and incudostapedial joint, an entirely different type of audiologic picture may exist. Where the physiologic fixation is limited to the tympanic membrane and lateral ossicles, the audiologic picture is still unclear.

The Fixed Malleus Syndrome is probably much more common than we realize. At the present time, the diagnosis is usually made when the air-bone gap exists and where the condition is primarily encountered during an exploratory tympanotomy. It may or may not accompany otosclerosis. It may be congenital or acquired. It may coexist with sensorineural hypacusis.

# PHYSIOLOGIC ASPECTS

Accurate studies of the acoustic sequelae and displacement characteristics of the human tympanic membrane in response to static pressure changes have not yet been made The problem of adapting techniques in use on the experimental animal to human studies is great but probably not insurmountable The recent report of Hoeft *et al* (1964) is of great interest in this regard They made direct measurements of the displacement of the guinea pig eardrum when acted upon by acoustic pressures (200 6000 cps) in the middle ear cavity Among their conclusions was the statement that the tympanic membrane may be represented as a flexible conical plate under slight tension and that there were two well defined nonlinearities namely hysteresis and a nonlinearity of the elastic properties of the ear It becomes obvious therefore that many changes in acoustic characteristic are possible as the result of varying degrees of increased stiffness of the tympanic membrane due to malleal head fixation

Moller (1965) measured the impedance of the tympanic membrane in the cat following malleal fixation by dental cement He states Below 2500 cps the impedance of the eardrum is high compared with that of the entire middle ear Above 3000 cps the impedance of the eardrum itself and that of the intact ear are of the same order of magnitude indicating that the eardrum vibrates in a way which permits only a fraction of its vibration to be transferred to the malleus Above this frequency the behavior of the eardrum is thus not the same as that of a rigid piston This is one reason why the sound transmission through the middle ear within this frequency range is not proportional to the inverse of the impedance at the eardrum as is the case at lower frequencies

In a series of important studies on sound transmission in the inner ear, Andersen *et al* (1963 *a* 1963 *b* 1964) contributed a great deal to our knowledge of the characteristics of sound transmission under the influence of artificially produced lesions in cadaver temporal bones In a recent paper Elpern *et al* (1965) report a clinical case of fixation of the head of the malleus which was dealt with by an arthrolysis produced by manubrial pressure and they report a significant improvement in hearing Unfortunately their report shows no bone conduction findings In their temporal bone experimental study they illustrate a rather dramatic low frequency transmission loss when the malleal head is fixed with cement They correctly point out that fixation lesions at sites other than the stapes footplate may produce a clinical picture simulating otosclerosis Several of the cases reported in this paper demonstrate that a completely

of the tympanum sheds much light on the interrelationships of the various ligaments and mucosal folds. This "mesentery" concept not only clarifies our thoughts on the spread of diseases within the middle ear, but it also begins to highlight the complex suspensory system of the incudomalleal mass. The pouches of von Troeltsch and Prussak and the notch of Rivinus must be added to the ligaments and to the tensor tympani muscle as we begin to conceptualize the anatomy of the "gear box" within the epitympanum.

The anterior malleal fold, the posterior malleal fold, and the suspensory ligament of the chorda tympani join with the malleal and incudal ligaments to create definitive compartments within the epitympanum. These compartments were ably described by Proctor.

Major variations in the height of the epitympanic roof and its lateral and antero-posterior dimensions may be easily observed in routine temporal bone dissections. In some specimens, the roof is fairly smooth, but in others bony septa project downward from the tegmen almost to the point of contact with the superior surfaces of malleus and incus.

A consideration of this complex supportive system makes it clear that there are a number of anatomical factors predisposing to malleal fixation. Thus, for example, anterior fixation could be due to persistence of a bony processus folianus anchored anteriorly, to calcification of the anterior ligament or anterior fold, or to anomalies of the supero-anterior aspect of the fibrous annulus. Superior fixation could be due to osseous fusion with tegmental bony septa or to calcification of the superior ligament. Such superior fixation could also be accompanied by incudal fixation because of calcification of the superior incudal fold. Similarly, the lateral ligament of the malleus and septa from the lateral attic wall could play a part in malleal head bony fixation.

in collaboration with F W Kranz (1928), and later modified into an electro-acoustic probe by Zollner (1951) and Thullen (1955), will undoubtedly enrich our armamentarium in dealing with observations of relative mobility of the tympanic membrane and its attached manubrium

Interestingly enough, the tensor tympani tendon did not seem to play a significant role in any of the cases in this series Particular attention was paid in every case to the possibility of either shortening or significant calcification of this tendon, but none was encountered Admittedly, this is a small series of clinical observations, and it is quite possible that limitation of tympanic membrane motion can be produced by lesions of the tendon of the tensor tympani muscle Indeed, this may be a fairly common lesion, the audiologic sequelae of which are still unclear.

The function of the tensor tympani in peripheral auditory processes has not been clarified, neither by impedance studies nor by audiologic techniques In a recent report, Clubb (1965) makes some very strong statements about the role of the tensor tympani in speech discrimination scores as well as in threshold pure-tone levels Local anesthetic sphenopalatine ganglion blocks and tensor tympani tendon "clippings" were carried out in a group of patients described as showing sensorineural loss and discrimination difficulties, a history of noise exposure, and retracted eardrums with prominent malleal short processes and folds Extraordinary improvements in discrimination are reported The report, however, gives no details of controls, nor are there details regarding calibration, test-retest studies, or possibilities of other audiologic variables We must await further studies along this line of investigation before attributing such dramatic sequelae to simple release of presumptive increased tension of the tensor tympani tendon, or to presumptive spasm of the tendon relieved by sphenopalatine ganglion anesthesia

fixed malleus may be accompanied by such a low frequency transmission loss which also may be superimposed upon a sloping, apparently sensori neural hypacusis curve with no air bone gap or only a suggestive slight air bone gap

Clinical physiological studies dealing with the middle ear have been focused largely on the fixed stapes and, in more recent years, upon the problem of ossicular discontinuity represented primarily by necrosis of the lenticular or long process of the incus. Thus, studies on impedance in the middle ear have dealt with the problem of the stapedius muscle (Feldman 1963 Feldman & Zwislocki 1965) and, to a lesser extent, with the tensor tympani muscle (Klockhoff, 1961) Attempts to make a diagnosis of either stapedial fixation or incus discontinuity are the primary present targets of impedance measurements, but no impedance studies dealing with specific fixation of the tympanic membrane secondary to malleal epitympanic fixation have been reported

Rigidity of the incudomalleal mass, when communicated to the stapes, produces lateral fixation of the stapes with the audiologic and other physiologic manifestations seen in otosclerosis What remains relatively unclear, however, is the group of physiologic sequelae presenting themselves in situations of incomplete fixation of the malleal head where there is relatively major stiffness of the incudomalleal mass and thus of the tympanic membrane but still some yielding in the relatively looser incudostapedial joint so that a true fixation of the stapes has not yet occurred.

The visualization of the relative mobilities of the tympanic membrane and the short process of the malleus on pneumatic otoscopic (Siegle) examination is a subject worthy of further exploration, preferably modified by some method of quantification.

In our clinic, we are now working with a preliminary model of an external auditory canal static pressure variation device with manometric control Visual observations of varying displacements of certain key landmarks (viz. umbo light reflex, short process) will be correlated with varying pressure changes.

The eventual possibility of obtaining direct impedance measurements from the tympanic membrane in the human are, indeed, hopeful. The vibrometer principle which Professor Y Ouchi has been studying in our laboratory at UCLA, as well as in his own laboratory in Tokyo, could well be the key to such successful direct impedance measurements. Such measurements would be of extreme importance in this syndrome and in general middle ear physiologic studies

Recent refinements in polytome roentgenography of the temporal bone will make X ray diagnosis of great value in precise delineation of relationships between the malleal head and the tegmen tympani. Such studies c[illegible]uld y[illegible] [illegible]tive preoperative evidence of epitympanic malleal head fixation.

Utilizati[illegible] [illegible]coustic probe first advanced by the late A. G. Pohlman

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# ETIOLOGIC ASPECTS

In 1860, Toynbee, in his monumental correlative study of otologic observations and autopsy dissections, described fixation of the ossicles as a cause of hearing loss, and he definitely differentiates fixation of the malleus and incus from fixation of the stapes

In 1885, Schwartze, in his work on surgical diseases of the ear, spoke about the problem of the "not so rare cases of isolated ankylosis of the malleal-incudal joint without concomitant ankylosis of the stapes" This is described under the heading of "Calcification of the Middle Ear Mucosa," which may well have been his description of what we now call tympanosclerosis

In his text published in 1909, Politzer discussed adhesions between the manubrium and promontory in which the malleus is fixed and again attributes this to band like adhesions comparable to those described by Toynbee He discussed methods of ossiculectomy and methods of removal of such adhesions

A number of contemporary writers (Goodhill, 1960, Guilford, 1963, Hough, 1963, House, 1963, Proctor, 1963 b, Hilding, 1965) have stressed the necessity of awareness of malleal head fixation, both in the area of tympanoplasty and as an unforeseen complication encountered during otosclerosis surgery

In a study of post-traumatic conductive hearing loss, Does & Bottema (1965) point out fixation of the malleal head in two cases requiring malleal neck transection In these instances, the malleal fixation appeared to be a consequence of skull fracture with probable fracture line extending through the epitympanic tegmental area

In his excellent study on post-inflammatory fixation of the malleus, Hilding (1965) discussed this problem, reporting one clinical case and another case encountered in a temporal bone specimen Ojala (1953), inhis illuminating study of temporal bone pathology, also reported a case of malleus fixation

A number of etiologic factors have been encountered in the production of the Fixed Malleus Syndrome These include tympanosclerosis, chronic granulomatous epitympanitis and mastoiditis, panossicular arthritis, cholesteatoma, osteoma replacing the superior malleal ligament, as well as a number of presumably idiopathic occurrences in which no specific pathologic lesion could be discerned The anatomical features described previously would easily explain the probability of calcification and fixation processes which are primarily due to anatomical variations Some specific primary

pathologic process may play a part in these supposedly idi[illegible] and further study may well elicit the existence of such a le[illegible] perhaps comparable to the osteogenetic process found in the [illegible] in otosclerosis The fact that such fixation may occur in [illegible] sensorineural lesions gives also a good deal of room for [illegible] proaches to etiology In most instances in this series, there [illegible] be a history of gradual progression in the severity of the fixati[illegible]

A valuable study of the histological characteristics of the [illegible] otosclerosis was made by Altmann (1965) in which he pointed [illegible] infrequent association of malleus fixation with otosclerosis H[illegible] stead of attributing the malleal lesion to malleal otosclerosis he [illegible] it as being a secondary formation resulting in an incudomalleal [illegible] In his analysis of the findings in 45 serially sectioned temporal b[illegible] patients suffering from labyrinthine otosclerosis with and with[illegible] ankylosis Altmann showed that the only pathological findin[illegible] ossicles were changes identified as of arthritic nature, limited to [illegible] cudomalleal joint In his interpretation this definitely was due [illegible] placement of the ossicular chain in patients with ankylosis and displace ment of the stapedial footplate He considers such malleal e[illegible] fixation as basically secondary and not due to a congenital anomaly [illegible] se or to otosclerosis in the malleal head per se

In the series of cases reported in this paper only one instance of malleal fixation was associated with definitive stapedial otosclerosis However, my colleagues and I have seen other cases illustrating the type of secondary incudomalleal fixation described by Altmann and agree completely with his thesis Only one case was reported (No 8) because it is specifically illustrative of several facets of such coexistent relationships

Surgical management will depend to some extent on the etiologic and pathologic factors involved Certain fundamental principles, however, may be stated for all cases A note of warning should be given in regard to *attempts to liberate the fixed malleal head by directing force with a blunt instrument to the manubrium* Such lysis of the malleal head can be obtained by force in this manner, *but it carries with it the great danger of* excessive pumping action through *the mobile* stapes to the cochlear perilymph, unless *preliminary* incudostapedial joint section is carried out Long-range results of such an arthrolysis are *relatively poor* from the point of view of refixation

Since the definitive diagnosis is usually made at the time of *surgery*, it is important *to mention* the differential palpation of the stapes vs the incus *It is very easy to* miss the diagnosis of *a mobile* stapes if attention is paid only to palpation via the *incudostapedial* joint or via the neck or crura

In many instances, as *mentioned* previously, the stapes may be fixed only by external *pressure from* the fixed incus It is *possible to demonstrate stapedial mobility in* such a case only by palpation with a blunt instrument upon the central *portion of the footplate* itself or on either the anterior or posterior pericrural *footplate regions* A dramatic difference will then be noted between such palpation *and palpation* via the capitulum or via the *incudostapedial joint It* is usually very easy to demonstrate a round window *reflex* on *pericrural palpation* in such cases

Step 1 *Lateral Atticotomy* Once the diagnosis of malleal fixation has *been made* by careful palpation, as described previously, a lateral atticotomy, produced by *removal of* a portion of the inferior aspect of the lateral epitympanic wall, is *necessary in order to* obtain adequate exposure of the incudomalleal joint This atticotomy is *easily accomplished*, either with the use of a cutting burr or with a small curet *It should be generous* enough to expose the *incudomalleal* joint throughout its extent and to allow the use of a knife *to sever the joint (Fig 1 A and B)*

Step 2 *Incudostapedial Joint Section* The next step *consists* of *incudostapedial joint section* with an *angulated 0 5* mm circumferential knife This *immediately liberates* the mobile *stapes* from transmitted incudal pressure in order to *protect* the cochlear *fluids from* violent excursions of *a pumping nature It also* liberates the incus sufficiently to allow for more precise palpation of the *incudomalleal joint* (*Fig* 2).

Step 3 *Incudomalleal Joint Section* The *incudomalleal joint* usually is *not fused and may* be sectioned with relative ease with a conventional

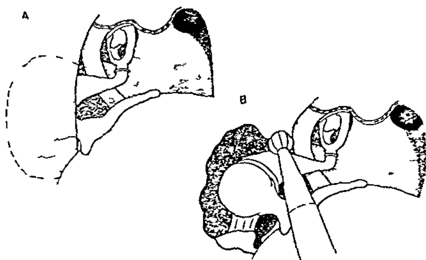



Fig 1 Lateral Atticotomy (A) Diagrammatic view of epitympanic area visualized before bone removal (B) Removal with cutting burr of a portion of the inferior aspect of the epitympanic wall to permit adequate exposure of the incudomalleal joint

1 mm angulated stapes knife In some cases however, the incudomalleal joint may be completely fused due to incudomalleal joint arthritis In such instances it may be necessary to employ a sharp cutting burr in an attempt to cut the joint In rare instances it may be necessary to leave the incudomalleal joint in situ within the epitympanum and amputate the long process of the incus in order to liberate the incudomalleal mass from the rest of the middle ear structures (Fig 3)

Step 4 *Incus Rotation and Removal* Following incudomalleal joint section the incus will become quite freely mobile and it is then relatively

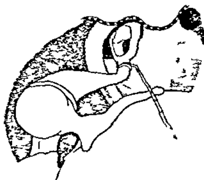


Fig 2

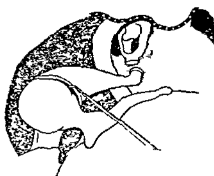


Fig 3

Fig 2 Incudostapedial joint section liberates the stapes from transmitted incudal pressure and thus protects the cochlear fluids

Fig 3 Incudomalleal joint section mobilizes the incus and prepares it for removal

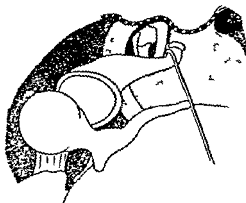


Fig 4

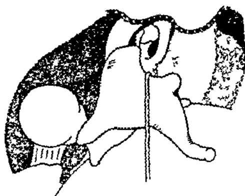


Fig 5

FIG 4 Rotation of incus away from the stapes

FIG 5 Delivery of incus from middle ear cavity

easy to lift it in a superior direction away from the stapes using a small hook or the angulated 0.5 mm circumferential knife After an elevation of approximately 45 degrees a rotation maneuver is begun which lowers the articulating surface of the incus away from the malleal articulation and then rotates the incus body out of its fossa incudis The incus now may be easily grasped with an alligator forceps and atraumatically removed from the tympanic cavity It is then placed in a shallow cup containing Ringer's solution *for subsequent remodeling into a hemi incus autograft* (Figs 4 5 and 6)

Step 5 *Malleal Neck Transection* This is the most crucial step in the entire surgical management It is necessary to completely separate the malleal manubrium from the malleal head in order to effectively liberate the manubrium and its attached tympanic membrane pars tensa It must be remembered that the primary pathologic lesion produced by the Fixed Malleus Syndrome is the complete immobilization of the tympanic membrane itself and this malleal neck transection is the only liberating maneuver This is accomplished by the careful use of a *1 or 1.5 mm cutting*

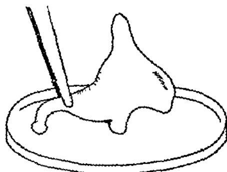
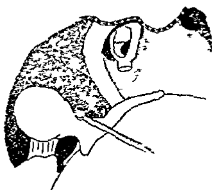


FIG 6 Placement of incus in Ringer's solution for subsequent remodeling



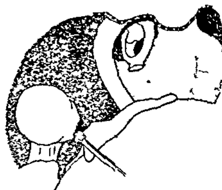




Fig 7 Cutting burr to malleal neck

Fig 8 Malleal neck transection completed allowing complete separation of the manubrium from the malleal head

burr Gelfoam pledgets saturated with Ringer s solution are placed in the medial aspect of the epitympanum and in the region of the manubrium for the purpose of protecting the mucosa and catching any bone dust that may accumulate It is necessary not only to cut through the bone of the malleal neck but also to completely sever all periosteal and submucosal tissue connections so that the manubrium is completely separated from the head, the neck actually being removed during the transection (Figs 7 and 8)

Step 6 *Management of Malleal Head* The malleal head may either be removed or left in situ in the epitympanum Ideally, it is best to remove the malleal head, but in some instances where the osteophytic connection with the epitympanic tegmen is extremely firm, removal may be hazardous and should be avoided (Fig 9 A and B)

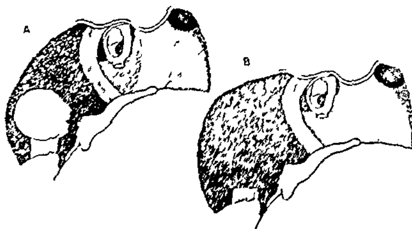



Fig 9 Mobile manubrium with (A) malleal head in situ (B) malleal head removed

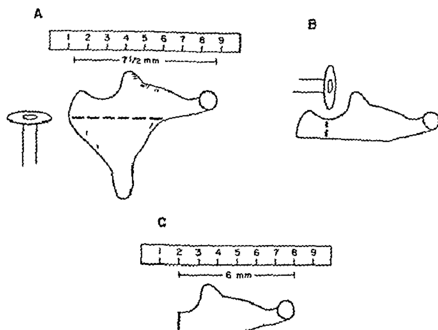



Fig 10 Hemi incus autograft prepared in regular (A) or shorter lengths (B, C) according to specific need

The re creation of a functioning mechanical transducer is achieved in this series of cases by the utilization of ossicular autografts in most instances Utilization of ossicular transplants has been reported by a number of writers in recent years Hall & Rytzner (1960) described repositioning of the incus Guilford (1965) has also used this technique to advantage Koide & Koike (1965) have advised ossicular repositioning to convert type III tympanoplasties into incudostapediopexies or malleolostapediopexy, and Rubinstein *et al* (1964) have advocated malleal transposition in certain middle ear problems *The specific needs in this group of cases appear to be best met by using a portion of the incus rather than the entire ossicle*

Step 7 *Preparation of Hemi-Incus Autograft* In most instances, the ossicular reconstruction following liberation of the tympanic membrane will be accomplished by the use of a hemi incus autograft created from the previously removed incus.[1] The incus is grasped with a fine toothed forceps, and a micro-saw attached to the regular handpiece is used to resect the short process and a portion of the body of the incus, thus leaving only the articular surface, the long process, and the lenticular process as a hemi-incus autograft *The average regular hemi incus measures approximately 7 5 to 8 mm in length* (Fig 10 A) In instances where a shorter hemi incus is necessary, the resection described above is combined with resection of the superior half of the articular surface, thus reducing

Hemi Incus Autograft in Reconstructive Tympanic Surgery" (in preparation) by Victor Goodhill M D

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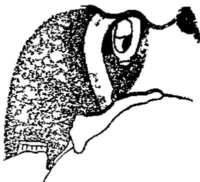




Fig 11

Fig 11 Elevation of manubrial muc per o graft

Fig 12 Hemi incus autograft from manubrium

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stoids bilaterally Audio-
4 A) Note the air bone
nduction curve and the

the hemi incus to approximately 5 to 6 mm
and C)

h a fixed malleus An
my were followed by
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In rare instances where it is impossible t
extensive incudomalleal osteoarthritis the
to the body, and a cartilage graft obtained fr
This technique is to be described in detail elsewh

of the air bone gap

Step 8 *Elevation of Manubrial Periosteum* Th
manubrium on its medial aspect is elevated from th
transection down to a point halfway between the n
prepares a very effective raw area for placement
cartilage autograft (Fig 11)

rs duration
findings slightly
oth drums were
X rays of both

Step 9 *Ossiculoplastic Reconstruction* A bed of
Gelfoam pledgets is prepared around the oval wi
region of the bared manubrium The autograft is then
so that its two ends are in excellent contact with the
most cases it is possible to create a V-shaped cut
graft so that its lateral aspect articulates with the
surface and the lenticular process usually fits directly
capitulum (Fig 12)

ss (Fig 15 A),
ly when peri
f otosclerosis
tapedial joint

If it is possible to preserve the posterior crus and
simple anterior crurectomy may suffice to allow good
as exemplified in Case No 1

d incus with
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In instances where the crura have been bent or where the
the manubrium and the stapes is awkward, a removal of the st
precedes the placement of the autograft In such instances

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* Tragal Cartilage Autograft In Reconstructive Tympanic Surgery by Victor Goodhill MD

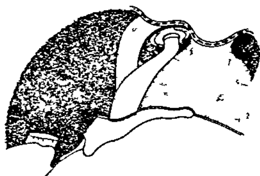


FIG 13 Hemi incus autograft from manubrium to mobile footplate

is placed directly between the manubrium and the mobile footplate, periosteum having been elevated from each receptive surface (Fig 13)

Following the accurate placement of the graft, the middle ear is closed in the usual manner and the canal packed as in normal poststapedectomy procedures

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# CASE REPORTS

## Case 1

This 54 year old man had a left hearing loss of one year duration. A sister had been operated by me for otosclerosis.

Examination revealed an intact and translucent left tympanic membrane. X rays showed well pneumatized normal mastoids bilaterally. Audiologic studies showed a left conductive loss (Fig. 14 A). Note the air bone gap but also note the slightly descending bone conduction curve and the bone conduction loss.

At surgery a mobile stapes was found along with a fixed malleus. An atticotomy, malleal neck transection and incudectomy were followed by an ossiculoplastic reconstruction utilizing a hemi incus between the manubrium and the footplate (Fig. 14 B).

This was followed audiologically by an overclosure of the air bone gap.

## Case 2

This 55 year old man had a left hearing loss of two years duration.

Otoscopic examination showed bilaterally symmetrical findings: slightly retracted but intact and mobile tympanic membranes. Both drums were mobile on pneumatic otoscopy and on Politzerization. X rays of both temporal bones showed infantile non pneumatized mastoids.

Audiologic examination revealed a conductive hearing loss (Fig. 15 A) and the diagnosis of left otosclerosis was made.

At surgery stapedial mobility could be demonstrated only when pericrural footplate palpation was used. There was no evidence of otosclerosis. The incus and malleus were both fixed. Following incudostapedial joint section an atticotomy revealed a completely fixed malleus and incus with a fused incudomalleal joint necessitating not only a malleal neck transection but also amputation of the long process of the incus. The body and short process of the incus remained rigidly fixed to the malleal head in the epitympanum. A cartilage graft from the tragus was tailored to fit between the bare manubrium and the bare footplate (Fig. 15 B).

This was followed postoperatively by a marked hearing gain in which there was average closure of the air bone gap with overclosure at 2000 and 4000 cps.

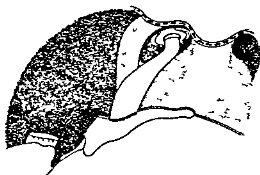


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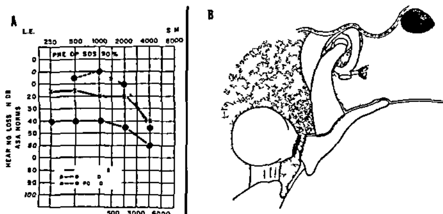


Fig 14 Case 1 (A) Pre and postoperat ve aud ogram show ing overclosure of tl e a r bone gap (B) Oss culoplastic reconstruction util z ng hem ncus autograft from manu brium to mobile footplate the poster or crus and stapedial tendon hav ng been pre served

## Case 3

This 43 year old male gave a history of bilateral hearing loss since childhood apparently following ear infections secondary to measles and scarlet fever

Initial examination showed left chronic otitis media perforata with mastoiditis The right tympanic membrane was intact but showed evi dence of a neomembrane closing a central perforation with a tympano sclerotic plaque anteriorly Mastoid X rays showed a good deal of sclerosis on the left with little pneumatization while the right was more pneuma tized Audiologic studies showed a bilateral conductive loss greater on the right Tuning fork tests showed negative Rinne responses on the right

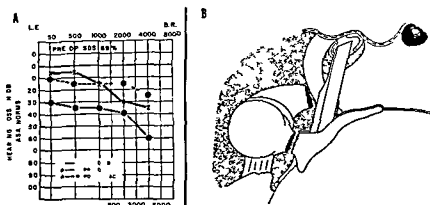


Fig 15 Case 2 (A) Pre and postoperative audiogram showing marked gain in hearing especially at 2000 and 4000 cps (B) Tragal cartilage autograft between manubrium and mob le footplate Note body of incus and malleal head left in situ due to rigid fixa t on of the entire incudomalleal mass

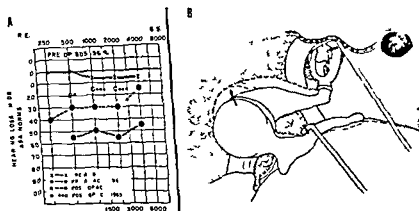



FIG 16 Case 3 (A) Air conduction gain following first surgical procedure on the right ear did not persist Improvement following second procedure was excellent but prognosis is guarded (B) Arthrolysis of the incudomalleal joint and fixed malleus by lateral pressure on the manubrium is illustrated

A left mastoidectomy removed extensive tympanosclerosis of the superficial non invasive type, allowing for a radical exenteration and a tympanoplasty type III Following this procedure, the ear healed nicely with a moderate gain in hearing Exploration of the right ear was advised

Right exploratory tympanotomy revealed apparent fixation of the entire ossicular chain by tympanosclerotic implants Incudostapedial joint section was followed by a stapedolysis with excellent stapes mobility and good round window reflex An arthrolysis of the incudomalleal joint and fixed malleus was performed by lateral pressure on the manubrium This was done slowly, resulting in complete restoration of mobility to the entire chain

Postoperative course was uneventful with an immediate excellent gain in hearing, however, the patient's hearing slowly regressed Therefore, 15 months later, a secondary stapedolysis and incudomalleal arthrolysis was performed, again followed by an excellent improvement in hearing (Fig 16 A and B)

This case illustrates (1) tympanosclerosis as a cause of malleus fixation, and (2) the possibility of dealing with malleal fixation by an arthrolysis, using lateral pressure to the manubrium While it did work, there was rather rapid regression due to refixation A secondary procedure was again sucessful, but the long term results remain doubtful It seems that malleal neck resection would have been the procedure of choice in this case

## CASE 4

This patient, a 55 year-old female, gave a history of a bilateral hearing loss of at least three years' duration The onset was gradual, but there was steady progression There was no otorrhea and no otalgia She had had a good deal of illness including hydronephrosis which required a right

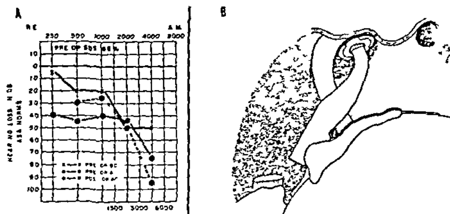



FIG 17 Case 4 (A) Postoperative air conduction level showed improved hearing at 500 and 1000 cps (B) Hemi incus autograft from upper manubrial margin to mobile footplate

nephrectomy She also had a bilateral salpingectomy, an appendectomy, and a thyroidectomy followed by an abdominal hysterectomy and bilateral oophorectomy She also required surgery for bilateral venous thromboses and had a skin cancer excised from the right side of her nose

Both tympanic membranes showed evidence of scarring of previous central perforations now closed by neomembranes X-rays of the mastoids showed evidence of bilateral periantral sclerosis with some halisteresis and rarefaction in each epitympanic area Audiologic studies revealed a bilateral marked conductive deficit with good discrimination in each ear There was a moderate high frequency bone conduction loss, slightly more marked on the right (Fig 17 A) In view of the history and findings, a right exploratory tympanotomy was advised under local anesthesia

At surgery an atrophic neomembrane was found invaginated beneath the annulus This was separated and removed The incus and malleus were found to be completely fixed to the lateral attic wall Following incudostapedial joint section, the stapedial footplate was found to be completely mobile with a good round window reflex External atticotomy was performed to expose the incudomalleal joint, which was found to be completely fixed to the epitympanum Following incudomalleal joint section, the incus was rotated and removed A malleal neck transection with a cutting burr was performed, and the malleal head was removed from the epitympanum The stapedial crura were removed The incus was cut through with a saw, and a hemi-incus formed consisting of a portion of body, long process, and lenticular process The manubrial periosteum was separated, denuding the manubrium medially, creating a bed for placement of the hemi-incus autograft between malleal manubrium and mobile stapedial denuded footplate A perichondral graft taken from the tragus was used to repair the previous central perforation The hemi-incus was kept in position with Gelfoam pledgets (Fig 17 B)

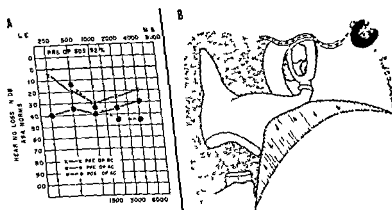



Fig 18 Case 5 (A) Early postoperative audiogram shows overclosure of the air bone gap in the low frequencies The 2000 and 4000 cycle levels were lower postoperatively (B) Type II tympanoplasty after total resection of malleus Perichondral graft in contact with incus

The ear healed uneventfully with an excellent improvement in hearing in the lower frequencies

This case illustrates the management of malleal fixation secondary to healed chronic otitis media What part did this patient's long history of many other diseases play in the etiology?

## Case 5

This patient, a 53 year-old female, had a series of bilateral ear infections accompanied by a progressive bilateral hearing loss A right mastoidectomy and tympanoplasty was performed with excellent results (intact graft and good hearing) She returned for care to the left ear when it began to suppurate following an acute respiratory infection, and the suppuration did not respond to antibiotic and local therapy Audiologic studies at that time showed a left conductive hypacusis( Fig 18 A)

At surgery, a low-grade granulomatous mastoiditis was encountered following a left endaural approach There was no cholesteatoma, and there was no tympanosclerosis The posterior bony canal wall was not disturbed, and the middle ear was explored The incudomalleal joint was found to be fixed, and there was no transmission between malleus and incus The stapes was mobile with an excellent round window reflex despite a good deal of peristapedial fibrosis The Eustachian tube was normal There was a small tympanosclerotic implant within the fibrous layer of the tympanic ... surrounding the central perforation, but it did not imm...

a cutting burr ... Hanu...

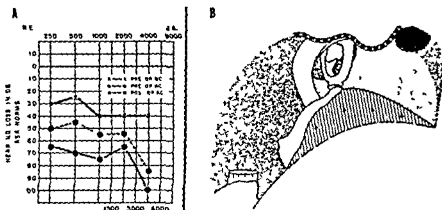


FIG 19 Case 6 (A) Pre- and postoperative levels (B) Manubrial Stapedial transposition creating myringostapediopexy (Tympanoplasty III)

brium of the malleus was then dissected away and removed along with its attached neck and short process, thus freeing the drum completely The malleal head was also removed, and then the incus became completely mobile The lenticular process was replaced on the stapedial capitulum Accordingly, it was possible to perform a type II tympanoplasty, utilizing a perichondral graft over the long process of the incus and under the peripheral posterior segment of tympanic membrane (Fig 18 B)

The postoperative course was uneventful with a significant gain in hearing in the lower frequencies on early postoperative audiograms

This case illustrates malleal fixation secondary to the epitympanic aspect of a granulomatous mastoiditis It also shows the possibility of solving the transmission problem through a type II tympanoplastic reconstruction

## CASE 6

This 67-year-old male patient was injured in an explosion approximately 45 years ago, following which he developed a right otorrhea and a severe right hearing loss The hearing, however, gradually improved, apparently returning to normal About five years ago, however, he began to notice a progressive hearing loss in both ears, with greater loss on the right side He had three surgical procedures performed on the right ear with no improvement in hearing He had no surgery to the left ear, in which he wore a hearing aid With the exception of hypertension which was under medical control, his general health was good

The right tympanic membrane was thickened, fibrotic, opaque and fixed, and there was evidence of a healed central perforation covered by a neomembrane The left tympanic membrane showed a large, dry central perforation Mastoid X-rays showed relatively poor pneumatization with marked sclerosis bilaterally Audiologic studies showed a bilateral conductive deficit with considerable bone conduction depression on the right (Fig 19 A) An exploratory right tympanotomy was advised

Right exploratory tympanotomy revealed advanced tympanic fibrosis. There was necrosis of the long process of the incus with the necrotic portion and its lenticular process still in contact with the stapes. The incus body and the head of the malleus were completely fixed in the epitympanum. Following incudostapedial joint section, the stapes was found to be quite mobile in spite of the peristapedial tympanic fibrosis. An external atticotomy with a cutting burr was performed, followed by section of the incudomalleal joint and rotation and removal of the incus from the epitympanum. The malleal head was completely fixed. A malleal neck transection with a cutting burr was performed, followed by removal of the malleal head from the epitympanum and resulting in a perfectly mobile malleal manubrium. The middle ear showed no evidence of tympanosclerosis or cholesteatoma. The medial aspect of the manubrium was denuded of its periosteum. It was then possible to mobilize and rotate the entire tympanomeatal membrane and place the malleal manubrium in contact with the exposed stapedial capitulum creating a myringostapediopexy (type III tympanoplasty) (Fig 19 B).

There was a significant gain in hearing postoperatively, making possible the transfer of the hearing aid from left to right ear. Future corrective surgery for the left ear is planned.

We see in this case a combination of incus necrosis (ossicular discontinuity) and malleal fixation (lateral ossicular and drum rigidity) accompanying a concomitant cochlear deficit due to repeated infections and to probable cochlear concussion by blast.

## Case 7

This 49 year old female had been aware of a severe left hearing loss since early childhood, apparently related to a number of ear infections. Within the last five years, she had also become aware of a right hearing loss of lesser degree.

On examination, the right tympanic membrane was found to be intact, translucent, mobile and only slightly scarred. The left tympanic membrane was only moderately mobile and showed a large healed central perforation covered by a thin neomembrane. A caloric vestibular test revealed normal responses bilaterally. X rays demonstrated highly pneumatic and well developed mastoids bilaterally with no sclerosis. Audiologic studies showed a moderate right sensorineural loss. On the left (Fig 20 A), there was a profound mixed loss with depressed bone conduction and an 80 dB air conduction level. A relatively high speech discrimination score of 80% on the left was surprising. Tuning fork tests showed negative Rinne responses on the left and positive responses on the right. Weber with all tuning forks was referred to the right.

Surgical exploration in 1963 revealed no evidence of an incus long process. There were no stapedial crura. A mobile footplate was present from

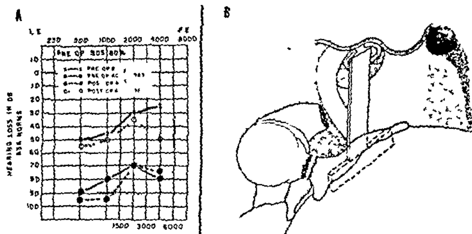



FIG 20 Case 7 (A) Pre- and postoperative air conduction levels show air-bone gap closure after the second operation (B) Cartilage T graft in place between reconstructed tympanic membrane and footplate

which a good round window reflex could be elicited No notation was made of the status of malleal mobility at the time of this surgical procedure, and it is not known if the malleus was mobile or not at this time A T-shaped cartilage strut was fashioned from tragal cartilage. Its medial aspect was placed on the mobile denuded footplate, and the T or arched portion of the autograft was placed laterally up against the very delicate neomembrane previously described and packed in place with Gelfoam (Fig 20 B)

Postoperatively, there was no improvement in hearing The neomembrane sloughed, however, and a large perforation appeared and persisted The cartilage graft remained intact and could be seen through the perforation Since the speech discrimination score continued to be surprisingly high in this ear (80%) in spite of the poor postoperative course, a subsequent exploration and an attempt at a myringoplasty was decided upon

The second surgical procedure in 1965 under general anesthesia utilizing an endaural incision revealed a very healthy mobile cartilage graft with a good blood supply, producing an excellent round window reflex (thus demonstrating excellent vitality of a cartilage autograft 18 months postoperatively) Further exploration at this operation, however, revealed complete fixation of the malleal manubrium due to the malleal head being rigidly fixed to the incudal body in the epitympanum Accordingly, a transection of the malleal neck was performed with a cutting burr, separating the manubrium from the malleal head and producing a mobile manubrium and tympanic membrane The drum remnant was de-epithelhated completely A canal wall free skin graft was obtained and placed over the T portion of the cartilage autograft and packed in place with Gelfoam over the denuded tympanic membrane annular region

The postoperative course was uneventful The drum healed completely,

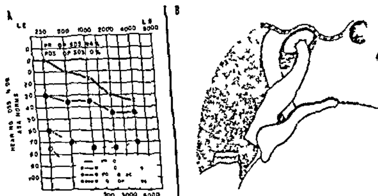


FIG 21 Case 8 (A) Severe sensorineural loss postoperatively (B) Hemi incus autograft from manubrium to oval window Note the Gelfoam pad over the oval window

and she has had a return of hearing to her preoperative bone conduction level with almost complete closure of the air bone gap

This case illustrates malleal fixation secondary to a circumscribed epitympanic infection without mastoiditis It also illustrates the viability and acoustic transmission qualities of a free cartilage autograft

CASE 8

This patient, a 72 year-old male, was first seen in 1963 with a complaint of gradual bilateral hearing loss of about ten years duration

On examination, both tympanic membranes were intact and mobile There were no significant nasopharyngeal findings Mastoid X rays were essentially negative Audiologic studies revealed a bilateral conductive deficit with a sloping high frequency loss bilaterally There was a good air-bone gap and a good speech discrimination score in each ear Tuning fork responses showed negative Rinnes at 128 and 256 bilaterally

A left exploratory tympanotomy in 1963 revealed complete fixation of the stapes by otosclerosis, but the incus and malleus were also fixed Anterior peribasal stapedolysis was followed by demonstration of good foot plate mobility An arthrolysis of the incudomalleal joint was then performed by pressure on the manubrium in a lateral direction This produced good motion and was followed by an excellent transmission from malleus through incus through the now mobile stapes with a good round window reflex There was no improvement in hearing subjectively, however, at the time of surgery

There was a very marked loss of hearing following the surgical procedure (Fig. 21 A), particularly in a lowered discrimination score which continued to vary and fluctuate over a period of two years This fluctuation ranged from 10% to 70% In view of the continued fluctuation and the relatively poor and close status of the left ear, a reexploration was deemed advisable

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